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THE
LARYNGOSCOPE.

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REVIEW OF AVAILABLE LITERATURE ON THE PHARYNX AND PHARYNGEAL SURGERY FOR 1952.

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ANATOMY.

In order to understand more thoroughly the development of the pharyngeal musculature, House[1] undertook the study of the hyoid bone in the albino rat. As he points out, complete knowledge of any muscle or group of muscles involves the question of origin and insertion which in turn entails knowledge of the skeletal structure. House found that the typical styloid process is absent in the albino; this is not surprising as absence of the styloid process is widespread among animals. The hyoid cartilaginous structure, which is composed of a body and two pairs of cornua, is described in detail. This article is the first of a series to be used as a background for a subsequent study of the development of pharyngeal musculature.

In a comprehensive article Brunner[2] describes the anatomy and physiology of the hypopharynx in minute detail and discusses the various theories of deglutition. He states that in normal persons the Valsalva experiment gives rise to spastic contraction of the cricopharyngeal muscle which forms a "cross-roll" at the posterior wall of the hypopharynx. This is

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analogous to Passavant's bar in the nasopharynx. The finding of a hypopharyngeal bar indicates spastic contraction of the cricopharyngeal muscle, and this is thought to be caused by the increased air pressure in the hypopharynx. Increased air pressure in the hypopharynx and a bolus of food in the hypopharynx have opposite effects upon the cricopharyngeal sphincter. Increased air pressure causes spastic contraction whereas a bolus of food causes relaxation of the sphincter and consequent opening of the cricopharyngeal sphincter.

Brunner[3] presents a discussion of the cricopharyngeal muscle under normal and pathologic conditions. He recalls that the inferior constrictor muscle is divided into an upper oblique portion and a lower portion, the cricopharyngeus. The upper portion balloons out during the Valsalva maneuver whereas the cricopharyngeus contracts, forming a "hypopharyngeal bar" which may be seen in lateral roentgenograms. Brunner thinks that the size of this hypopharyngeal bar is an indication of the degree of excitability of the cricopharyngeus muscle. A spontaneous bar is seen in certain nervous disorders. The bar is frequently large in patients with a lesion near the hypopharynx; the bar is not abnormal in patients with laryngeal nerve paralysis.

PATHOLOGY.

Davalos[4] describes the Waldeyer's ring, its extension and significance and discusses its relationship with other structures. He points out that whereas in children, the lymphoid tissue is frequently hypertrophied, this does not always mean infection. Hypertrophied lymphoid tissue occurring in children should always be studied carefully before concluding that the tonsils and adenoids should be removed. Allergic manifestations are responsible for clinical changes in the appearance of the lymphoid tissue and also frequently produce asthmatic symptoms. Davalos enumerates the most frequent symptoms produced by allergy in children and urges that no child with such symptoms be submitted to tonsillectomy. Following tonsillectomy, too often symptoms persist simply because allergic investigation was not undertaken

prior to the operation. Davalos attempts to warn otolaryngologists that the presence of tonsils and adenoids is no indication for operation. His advice merits attention, and a more careful study of the cause of hypertrophy of the lymphoid tissue of the pharynx and nasopharynx would lead to fewer disappointments following tonsillectomies and adenoidectomies.

DIAGNOSIS.

Rhinometry is a method of determining the patency of the nasal passage. Various indirect and direct methods of measurement of nasal air flow, pressure and volume have been devised. Staksted[5] has devised a method using a nasal plug, water manometer and an electrocardiograph by which the permeability of the nasal passage can be determined and compared with normal values. This has been used in children with enlarged adenoids both before and after adenoidectomy. No correlation between the amount of adenoid tissue removed and the amount of nasal obstruction could be demonstrated.

Referring to the nasopharynx as the "neglected area," Theobald[6] points out that there are few specialties in medicine for which the nasopharynx is completely without interest. To the neurologist it is an area where involvement is capable of producing an interesting array of neural involvement from the first to the twelfth cranial nerves. To the ophthalmologist involvement of the third and sixth cranial nerves not infrequently presents bizarre ocular symptoms. To the surgeon who is concerned with swellings of the neck, the nasopharynx, as the site of primary neoplastic diseases, must ever be borne in mind. To the otolaryngologist the region of the epipharynx represents the source of postnasal secretions, recurrent sore throats, cough, earache, fever and hemorrhage in addition to those conditions already mentioned. Routine examination of the nasopharynx is so simple a procedure that failure of examination constitutes neglect. In the examination of this area the mirror, pharyngoscope, palate elevator and Yankauer speculum should be

utilized. Interesting cases including pharyngeal bursa (Thornwaldt's bursa), tuberculosis, lymphoid hyperplasia and their treatment are discussed.

Carrell[7] describes a technique for photographing the movements of the tongue, soft palate and pharynx in speech, consisting of taking motion pictures of the area through a fluoroscopic screen with a fast lens motion picture camera. He believes that the possible clinical applications may well ultimately include cinefluorography as a routine diagnostic aid in determining the status of velopharyngeal function, evaluation of the results of variations in surgical techniques, and aiding in decisions such as repositioning of the soft palate; however, he points out that the clinical applications may be limited by the cost of cinefluorographic instrumentation and the specialized personnel required.

Lachapele and associates[8] present a case to demonstrate the value of tomography in the diagnosis of diseases of the hypopharynx. The patient had a right subclavicular adenopathy which biopsy showed to be an atypical epithelioma. Examination of the pharynx yielded negative results. Frontal pharyngolaryngeal tomography localized the tumor.

DISEASES.

At the meeting of the Société de Laryngologie des Hospitaux de Paris, March 13, 1951, Leroux-Robert and Robert[9] presented the case of a patient, aged 73 years, with Hodgkin's disease in whom the initial manifestations were in the hypopharynx. They were able to find only one other analogous case in the literature.

Gamard[10] calls attention to a little known disease, pharyngomycosis, which George Laurens defined as an essentially benign, parasitic and contagious angina characterized by the presence of white spots on the surface of the tonsils, wall of the pharynx and base of the tongue, containing a fungus, Leptotrichiabuccalis. The condition may occur in those who were not vaccinated for diphtheria or in whom vaccination was incomplete. According to Gamard, once the disease has been encountered it is easily recognized. He considers specific

treatment to be Mycodecyl applied in powder form or in solution. An illustrative case in a 13-year-old child is included.

Hallinger and associates[11] present an excellent although brief discussion of the differential diagnosis and management of hoarseness in children. Diagnostic measures include a careful history, mirror or direct laryngoscopy, and examination of the heart and lungs. Lateral roentgenograms of the neck showing laryngeal, pharyngeal and tracheal structures are often helpful. A discussion of the treatment of vocal nodules and papillomas of the larynx in children is included.

Korkis and Stein[12] consider white tuberculosis of the larynx a common clinical entity in connection with pulmonary tuberculosis; involvement of the pharynx alone is, on the other hand, an unusual complication. They report the case of a patient complaining of irritation of the throat in whom stagnation of mucus was seen in the left pyriform fossa. Direct laryngoscopy disclosed an ulceration with evidences of granulation tissue in the pyriform fossa. A roentgenogram of the chest revealed bilateral pulmonary tuberculosis. Good results were obtained from administration of streptomycin. Granulation tissue and ulceration of the pyriform fossa are always suggestive of the possibility of some malignancy, and this was ruled out by biopsy.

Jacod[13] presents in detail a case to illustrate the clinical picture of paresthesias and chronic pharyngeal manifestations in patients with gouty rheumatism. He believes that the best internal medication for this condition is piperazine. It must be given continuously over a long time in sufficient dosage to be effective.

In an interesting article concerning rational therapy directed to the nose, throat and ear, Fox[14] calls attention to the fact that local therapy directed toward infections of the tonsils and pharynx has long been dominated by the use of gargles, which have been shown to have almost no effect on the areas under consideration. Frequently, the ineffectuality of gargles can be demonstrated by having a patient

gargle with a solution of methylene blue. Examination of the throat immediately afterwards reveals stain on the hard and soft palates, the dorsum of the tongue, the anterior pillars and the base of the uvula, but the tonsillar surfaces, posterior pharyngeal wall and even the main body of the uvula are unstained. The use of lozenges is more effective in spreading the desired medication over the affected areas and Fox even prefers sulfathiazol chewing gum to use of lozenges. The use of troches of penicillin, aureomycin, chloromycetin and to a lesser extent Bacitracin often results in an annoying glossitis and stomatitis which patients consider worse than the original sore throat. We are in thorough accord with this last statement, for it has been our experience that in a large percentage of patients using antibiotic troches severe stomatitis develops which time alone will heal.

Riggs[15] calls attention to the importance of benign nasopharyngeal disease as the cause of many symptoms, the most common being postnasal discharge, occipital headaches, recurrent sore throat and crusting. The most common pathologic conditions existing are nasopharyngeal abscess, chronic inflammation of lymphoid tissue and Thornwaldt's disease. Complete examination of the nasopharynx including the use of the Yankauer speculum is advocated. Many patients were relieved by local application of silver nitrate to the diseased area. Riggs believes that surgical removal of the diseased nasopharyngeal area is best accomplished under local anesthesia and describes his technique in detail. Postoperative hemorrhage has not been a problem in his hands.

DIVERTICULA.

Welti[16] makes a plea for the two-stage diverticulectomy not only for extremely large diverticula but also for the small ones. His discussion is based on seven cases. He admits that the two-stage procedure may be complicated by cellulitis and mediastinitis, which are always to be feared despite the advent of the antibiotics; however, he points out that by means of a two-stage operation it is possible to remove the entire sac up to its neck without danger and to place the

ligatures in an area formed only of mucus, thus eliminating the risk of recurrence. He believes that the two-stage procedure deserves renewed consideration because of its innocuousness, the tranquillity which it gives the surgeon, and the complete cure which it provides.

Gautier and Tacquet[17] report the case of a woman, aged 62 years, who had a voluminous diverticulum of the hypopharynx, measuring 15 cm. by 7 cm. by 4 cm., which caused almost complete dysphagia, regurgitation, considerable loss of weight and edema of the ankles, face and hands. Surgical excision in one stage resulted in complete cure.

Rose[18] reports the case of a diverticulum in a 69-year-old man. Because of its unusual size, the diverticulum, which measured 7.5 by 7.5 by 5 cm., extended into the mediastinum and obstructed the esophagus. It was removed in one stage through a transverse incision in the neck. Since the operation the patient has had no dysphagia. A small residual pouch remained, but it has not increased in size in the two years since the operation and is, therefore, not believed to be a recurrence.

Following a brief review of the symptomatology and pathogenesis of pharyngoesophageal diverticula, James[19] describes the technique of Sweet for a one-stage operation for correction of these lesions. In seven patients in whom this operation was performed the results were uniformly satisfactory, and there were no complications.

Conole and D'Angelo[20] report a case in which during surgical removal of a pharyngoesophageal diverticulum the patient experienced a violent paroxysm of coughing followed by increasing respiratory difficulty. The operation was completed, the patient's distress became more acute, and severe abdominal distention developed. The peritoneal cavity was opened via a lateral rectus incision and a great gush of air emitted. The patient immediately improved. Conole and D'Angelo postulated that air entered the mediastinum through the incision in the neck, and passed through the hiatus of the diaphragm which acted as a ball valve producing a tension pneumoperitoneum.

In an interesting article Bjork[21] reports that he encountered hypopharyngeal diverticula in four members of the same family, a woman and her three daughters. Obviously there was a familial tendency to hypopharyngeal diverticulum in these cases. The hereditary tendency can scarcely be explained on any other basis than as an anatomic peculiarity; that is, an exceptional weakness in the posterior wall of the hypopharynx. From a review of the literature Bjork concluded that there are many conflicting opinions on the pathogenesis of hypopharyngeal diverticulum. He quotes from the observations and theories expressed by Negus, Dunhill and Vinson. He is of the opinion that if the posterior wall of the hypopharynx is particularly weak, due to a hereditary anatomic peculiarity, a physiologic "deglutition pressure" suffices to cause the formation of a pouch.

In a most interesting but short report Morris[22] calls attention to the fact that pharyngeal diverticula of traumatic origin are common among the habitual convicts in the central and united provinces of India. These diverticula are self-inflicted, generally unilateral and serve to hide coins, jewelry or precious stones. Some diverticula may hold up to 15 or 20 rupees. These diverticula are made by using a piece of lead about the size of a pigeon egg which is placed in the tonsillar fossa at bed time. A string passed through a small hole in the lead and fastened to the ear will prevent any slippage into the esophagus. The lead, which causes ulceration and destruction of the tonsil, is removed each day and replaced at night for 15 or 20 days, when a good pouch is formed. This is enlarged by finger massage and objects are placed within the pouch for safe keeping.

Realizing that a great deal has been written on pharyngeal diverticula, Atkinson[23] discusses only the rarer types, and attempts to correlate these with better known varieties of pharyngeal pouches and with certain anatomic and embryologic factors. Pharyngeal diverticulum of the pulsion type is the most common. The lateral pouches are usually attributed to developmental errors. The anatomy and embryology are well discussed, and the mode of development of posterior

pulsion diverticulum is well described. Lateral pouches may be produced by repeated inflation of the pharynx by air, or deliberately by digital distention, or they may be of developmental origin. Anatomic dissection of the lateral and posterior pharyngeal wall discloses weak areas between the middle and upper and lower constrictors. Atkinson relates the case of an elderly man who formed the habit of inflating his ears by holding his nose and blowing hard. Eventually a pulsion diverticulum and bilateral well defined pharyngoceles developed. Similar conditions occur in musical instrumentalists and glass blowers. As a rule these traumatic pouches are made between the palatoglossus and the palatopharyngeus, extending laterally between the stylopharyngeus and styloglossus beneath the lower edge of the superior constrictor.

In a discussion of pulsion diverticulum of the hypopharynx, Knight[24] describes the etiology and symptoms. To correct these conditions he prefers the one-stage operation in conjunction with esophagoscopic assistance, although some general surgeons continue to use the cumbersome, prolonged two-stage procedure in which the sac is first exteriorized and anchored in the wound near the skin. Ten to fourteen days later it is excised and the wound permitted to close by secondary intention. Knight is of the opinion that this latter method invites infection and other complications. The one-stage operation with the introduction of the esophagoscope has proved its worth over a period of years.

In a clinical lecture delivered in the Medical School, Guy's Hospital, Eckhoff[25] presents a standard textbook discussion of pharyngeal diverticulum.

FOREIGN BODIES.

In an interesting article on emergencies in otolaryngology Proetz[26] warns the general practitioner of the dangers of foreign bodies lodging in the pharynx. He presents an interesting case of a tack embedded in the dorsum of the tongue. The first principle in dealing with foreign bodies in the hypopharynx is to analyze the problem at hand and to refrain from attempted removal until necessary armamentarium is

available. It is best to have the patient in the supine position with the head slightly below the level of the body. In this position the foreign body cannot slip away and enter the tracheo-esophageal passages. Helpful hints are given for the handling of these cases.

HEMORRHAGE.

Harkins[27] emphasizes the importance of prompt, careful investigation in all patients bleeding from the nose and throat. The bleeding source should be located and methods for controlling it promptly instituted. Harkins warns against use of an inhalation anesthetic in children who have been bleeding for a considerable time until adequate transfusions of whole blood have been given and the hemoglobin level has been adequately determined. For slight oozing in the tonsillar fossae after removal of clots, topical application of thrombin is advocated. For extensive hemorrhage from the nose and throat, oxidized cellulose dressings are effective, easy to apply and readily absorbed. Harkins expresses the belief that the combination of vitamin C and vitamin K "seems to give the patient convalescing from a tonsillectomy and adenoidectomy the best chance to recover without complicating bleeding."

Olson[28] reports a case of epistaxis because of the unusual location of the hemorrhage. The patient was a 26-year-old woman who had had an attack of profuse bleeding from the nose and mouth which could not be controlled by packing of both nasal chambers with vaseline gauze. At operation the bleeding point was located on what appeared to be the left lateral or superior wall of the nasopharynx, or both. A nasopharyngeal pack and repacking both chambers with vaseline gauze controlled the bleeding.

At the meeting of the Société de Laryngologie des Hospitaux de Paris, July 2, 1951, Bourgeois[29] presented a rare case of profuse hemorrhage in an infant who had the Claude Bernard-Horner syndrome associated with hemiplegia. Otorhinolaryngologic examination revealed a lateral pharyngeal abscess, excision of which released a small amount of pus.

This was immediately followed by such profuse hemorrhage that the infant collapsed. The bleeding, however, was controlled and the infant responded favorably to supportive measures. During the ensuing months the Bernard-Horner syndrome and hemiplegia improved, but the infant had several more attacks of hemorrhage. Ligation of the primary carotid artery following the last attack of bleeding resulted in cure. Bourgeois was not able to find any reports in the literature of retropharyngeal abscesses associated with such profuse hemorrhages.

IRRADIATION.

Rennaes[30] reports a series of 130 cases of hypopharyngeal tumors treated by radiotherapy. In all but 13 the diagnosis was confirmed by histologic examination. The tumors are classified into extrinsic and postcricoid lesions. The subjective symptoms and technique of treatment are described and a discussion summarizes the results obtained and forces the author to the conclusion that radiotherapy in effect is mainly palliative. A five year cure following Roentgen treatment was obtained in 6 per cent of the cases, and from observation it was concluded that the extrinsic type of lesion responded more favorably to radiation than the postcricoid.

Loch and Fischer[31] examined 85 patients who had received nasopharyngeal radium application from three to 29 years previously. There was no evidence of detrimental effect from this irradiation in a single case. There was a small amount of lymphoid tissue in the nasopharynx in two-thirds of the cases. The long term results as to Eustachian tube patency were encouraging. They consider the proper plan of treatment to be application of 12 minutes to each Eustachian tube orifice at two-week intervals with the Crow applicator.

SURGERY.

In a brief article Podvinec[32] comments on voice production and the importance of first acquiring and mastering the requisite consonant sounds. The production of these sounds is discussed, and it is agreed that firm closure of the nasopha-

ryngeal opening by the soft palate is absolutely necessary. If the palate fails to close the nasopharyngeal opening, there is perceptible impairment of speech, and the voice is lacking in resonance and musical quality. Thus, open rhinophonia cannot be regarded simply as the result of escaping air into the nasopharynx during speech; it must also be considered as a definite handicap in voice production. It is, therefore, obvious that in cases where open rhinophonia is the result of the velum of the palate's being too short, cicatrized or of reduced mobility, good speech can be obtained by reducing the size of the passage between the oropharynx and the nasopharyngeal cavity. Podvinec has devised a method of pharyngoplasty, the purpose of which is to create a new partition in the patient's pharynx located near the base of the tongue, which according to his reports, is highly successful.

BENIGN TUMORS.

Following the brief discussion of neurogenic tumors of the pharynx Varvin[33] describes the case of a woman with a small pedunculated tumor above and behind the right tonsil, which was successfully removed, and proved to be a neurofibroma.

According to Heck and McNaught[34] juvenile fibromas, which are highly vascular, benign neoplasms occurring in the nasopharynx of pubescent males, probably arise from the perichondrium of a cartilaginous plate between the body of the sphenoid and the basi-occiput. Symptoms include hemorrhage, nasal obstruction and facial deformity. Two cases are presented in which surgical removal was affected perorally by a palate splitting procedure. The advantage of this approach is that it leaves an essentially normal structure as contrasted with the anatomic disturbance remaining after the transantral approach.

Attention has been called frequently to the occurrence of lesions in the tonsils and nasal cavities in patients with sarcoidosis, but little interest has been shown in the occurrence of these lesions in the nasopharynx. Larsson[35] reports 11 cases of microscopically proved lesions in the nasopharynx in association with sarcoidosis. The lesions were circum-

scribed, lobulated, reddish yellow masses in the adenoid area. In some cases they were large enough to cause discomfort and nasal obstruction. Those lesions producing symptoms were treated by fractional irradiation with uniform success.

Parnell, Alexander and Robertson[36] report an interesting and unsual case of pharyngeal tumor. Seven months before, during a severe coughing spell, an elongated red mass was coughed up and lay on the tongue and protruded well beyond the front teeth. In order to regain his breath the patient had to swallow the tumor, which was described as red, beefy and non-tender. There was no hemoptysis, dysphagia or pain in the neck or throat. It was determined that this growth represented a pedunculated tumor of the esopharynx attached to a ridge of the aryepiglottic-fold and because of an increased vascular supply it was thought best to remove it by an external incision. This was successfully accomplished, and a pathologic diagnosis of "massive development of papilloma of pharynx" was rendered by the pathologist.

This is an interesting case not unlike other rather similar cases which were called pedunculated pharyngeal lipomas. The pathologist found it difficult to establish a diagnosis which would encompass all the findings.

Jones[37] reminds us that benign tumors of the pharynx are comparatively rare and that papillomas, cysts and lipomas are the most common types. Jones presents a case of a 48-year-old man who spoke as though he had a large plum in his mouth. He complained chiefly of severe snoring, hoarseness and dysphagia. Pharyngeal examination revealed a smooth, soft tumor in the hypopharynx, covered with mucous membrane. Surgical removal presented no particular problem, as the tumor was attached by a pedicle to the right aryepiglottic fold; pathologic examination proved this to be a lipoma.

Penfold[38] reports a fatal case of lipoma originating in the hypopharynx not only because such tumors are infrequently encountered but also because they rarely are a cause of death. The patient was a 58-year-old woman who was found one morning with a tumor protruding fully four inches from the

edge of her mouth. She was unable to speak, had dyspnea and severe cyanosis and shortly afterwards died. Autopsy revealed that the tumor was attached to the anterior wall of the pharynx in the cricopharyngeal region. The patient had no previous symptoms indicating existence of the tumor. Penfold concluded from a review of the literature that lipomas of the hypopharynx and upper part of the esophagus produce few or no symptoms until they become real large. Invariably these tumors are pedunculated.

Because of the rarity of neurilemmomas occurring in the pharynx, Hanley and Davol[39] report the case of a 24-year-old white woman in whom routine examination disclosed a swelling of the posterior pharyngeal wall about the size of a bantam hen egg, which was firm, non-tender, non-fluctuant and asymptomatic. A mild Horner's syndrome existed on the right side, indicating probably that the growth arose from the cervical sympathetic chain. The tumor was successfully removed transorally. These tumors usually arise from the sheath of the peripheral nerve and unlike neurofibromas are usually single and do not undergo malignant change.

Somers[40] presents an analysis of 29 cases of neurogenic tumors occurring in the pharynx derived from elements of the peripheral portions of the nervous system collected from the literature and reports a case of neurilemmoma of the pharynx in a man 20 years old. The tumor was associated with left mandibular swelling and was diagnosed as a retropharyngeal abscess at one time and cervical tuberculosis at another. The patient was operated upon, and a tumor was removed from the pharynx. The pathologic report was benign neurofibroma. The patient made an uneventful recovery.

Som and Wolf[41] emphasize the fact that whereas benign neoplasms of the hypopharynx occur infrequently, they may grow to relatively large dimensions without being detected. The commonest of these is the fibrolipoma, which may be single or multiple, sessile or pedunculated. The pedunculated ones are potentially hazardous. One such case is reported in which sudden dyspnea developed; indirect laryngoscopy

disclosed a large, rounded, smooth tumor covered by intact mucosa overhanging the left arytenoid. Suspension laryngoscopy was utilized to expose and visualize the tumor more adequately. The tumor was attached to the posterior portion of the left arytenoid and was removed by snare and diathermy. A similar case formed a second report. Fibrolipomas may remain dormant until they attain good size and then produce alarming symptoms of dyspnea and dysphagia. Such tumors attached to the posterior portion of the larynx are best removed by suspension laryngoscopy.

A panel discussion in the Cancer Bulletin[42] states that fibromatous tumors occurring in the nasopharynx occasionally present real problems. The high vascularity of these tumors may result in fatal hemorrhage. The growth is not encapsulated; therefore, like other tumors lacking a surrounding membrane, a nasopharyngeal fibroma can destroy bone, and it may even invade the base of the skull. These tumors occur almost exclusively in young people. Nasal obstruction and epistaxis are the most frequent symptoms. The examination presents no problem, but when a nasopharyngeal fibroma is found, biopsy may be accompanied by severe hemorrhage. Regardless of this fact, biopsy is always imperative. The panelists believe that if the diagnosis is made early, the growth can often be eradicated by surgical excision. Radiotherapy may be employed preoperatively to reduce the over-abundant blood supply of the growth. The panelists agreed that a nasopharyngeal fibroma does not usually regress spontaneously as the patient becomes older. If treatment is adequate and well executed, the prognosis is excellent. Mention is made in this article that the panel discussed the advisability of radium and radar implantation on repeated occasions in an effort to reduce not only the vascularity but also the size of the tumor, thereby facilitating the surgical procedure.

At the meeting of the Société de Laryngologie des Hospitaux de Paris, Feb. 15, 1951, Millard[43] presented a case of mixed salivary tumor of the retropharyngeal space because of its rarity; according to him only about a dozen cases

have appeared in the literature. The patient complained of pharyngeal discomfort and some difficulty in swallowing. Examination revealed a tumor behind the pharynx on the left side. Following excision of the tumor radiotherapy was employed for some weeks. A normal result was obtained.

MALIGNANT TUMORS.

At the meeting of the Société de Laryngologie des Hopitaux de Paris, July 2, 1951, Bourgeois[44] presented a patient in whom one year before half of the pharynx and larynx had been removed because of a small neoplasm in the posterior aspect of the arytenoid. Radiation therapy was begun one month postoperatively. About eight months postoperatively the patient complained of an earache on the operated side, which Bourgeois attributed to a subclavicular ganglionic recurrence, since the local result was perfect. At the time the case was presented the patient was able to breathe and swallow without difficulty.

Piquet and Tupin[45] discuss the clinical manifestations, pathology, diagnosis and treatment of malignant granulomas of the face and pharynx and report a fatal case in a patient 43 years old. They point out that current methods of treatment have been generally unsuccessful. At present early radiation and surgical excision offer the best methods of management.

Nakagawa[46] presents an analysis of the records of 2,247 patients seen in the Radiological Therapy Department of the Japanese Cancer Hospital during the three year period ending Oct. 1, 1949. Neoplasms of the oral cavity and pharynx were found in 204 of these patients. There were 167 carcinomas of the oral cavity and 27 sarcomas. Of the 56 tumors of the pharynx 21 were carcinomas and 30 were sarcomas. His analysis thus reveals that carcinoma was encountered much more often than sarcoma in the oral cavity whereas in the pharynx there were three sarcomas for every carcinoma.

Edgerton[47] states that in many cases it is now possible to reconstruct the oral cavity or pharynx at the time of surgi-

cal excision of malignant or radio-necrotic ulcers. In extrinsic carcinomas of the larynx, hypopharynx or even the cervical esophagus it is usually necessary, in addition to removal of the larynx, to excise the greater part of the circumference of the lower pharynx and cervical esophagus. This recent return to more extensive radical surgical excision in the treatment of tumors of the head and neck has made the plastic procedure described by Harold Wookey acceptable in many clinics; however, Edgerton's experience with this method has been very disappointing. Four cases of reconstruction of the cervical esophagus and hypopharynx are described in detail. Free split-thickness skin grafts, which serve excellently to replace the lining segments of the esophagus and pharynx were employed. They are applied by fastening the grafts to a metallic mesh, which in turn serves as a stent or an elastic mold. According to Edgerton these patients not only partake of a regular diet, but in many of them acceptable esophageal voices develop. Such fundamental work is sound, and as it develops, more and more cases of carcinoma of the hypopharynx are destined to more complete cure and rehabilitation.

In reporting the results of Roentgen therapy in eight cases of carcinoma of the posterior pharyngeal wall Caulk[48] states that when the lesion originated between the levels of the arytenoid cartilage and the hyoid bone no cures were obtained. The conclusion was that advanced carcinoma of the hypopharynx, regardless of site of origin, offers a poor prognosis from the standpoint of Roentgen therapy.

Priest[49] presents an interesting discussion of plasma cell tumors of the nose, nasopharynx, oropharynx, hypopharynx and larynx. These tumors occur quite frequently in bone marrow constituting one of the histologic types of multiple myeloma. The case reported presented masses from the posterior choanae, the posterior pharyngeal wall and the larynx. Histologic examination proved these to be plasmocytoma. The inherent uncertainty of the prognosis substantiates the advice to consider plasmocytoma malignant and it should be treated as such. Priest states that as long as the

tumor is localized and confined to soft tissue, a clinical cure may be obtained by surgical removal or irradiation or both. On the other hand, therapeutic measures are usually useless if applied after the tumor has either locally invaded bone structures or has spread to lymph nodes or to the skeleton.

Ewing and Foote[50] tell us that multiple myeloma is a progressive, fatal disease characterized by the presence of widely scattered tumors, mainly in the bone marrow of those adult bones in which hemopoiesis is most active. Occasionally, single osteolytic lesions are encountered in bone that has the radiologic and histologic features of any one of the tumors of the multiple type. On occasions this same type of tumor is encountered in soft tissues quite separate from bone and by far the most frequent location of the solitary plasma-cell myelomas are in the upper air passages. Ewing and Foote reviewed hospital records of 27 cases of solitary plasmocytomas all arising in the nasal cavities or sinuses.

For purposes of treatment these tumors were grouped into three classifications. The treatment and results obtained from this type therapy is reported. Seven of the nine patients in Group 1 were dead at the end of two years. Group 1 included patients with local manifestations of a systemic disease. Group 2 consisted of those patients treated surgically occasionally in conjunction with irradiation. Two patients remain well after four and one-half years. In Group 3 the immediate result from irradiation was quite dramatic; however, only five patients out of 11 have survived two years. Ewing and Foote are forced to the conclusion that it is unsafe to regard any cases of solitary plasmocytoma as noncancerous. They believe that all cases of plasmocytoma are potentially, and in fact often, malignant and conclude by agreeing that there is no sharp line of demarcation between localized, benign plasmocytoma on the one hand, and the malignant fatal multiple myelomas on the other. This is an excellent article dealing with a rather rare type of tumor, and the method of treatment is well worth reviewing.

In an excellent discussion of the problem of carcinoma of the pyriform fossa and postcricoid carcinoma by several

English authorities[51] all are in accord that the treatment of choice in both types of cases is surgical excision of the lesion and primary and secondary lymphatics. This consists in pharyngolaryngectomy and block dissection of one or both sides of the neck. There is some controversy over the type of esophageal closure that should be done. Each discussor presents his views and describes his technique in some detail.

Raven[52], in an all too short article, describes his method of handling a malignant growth on the lateral wall of the oropharynx which involved the tonsil, fauces, soft palate, and base of the tongue; metastases were also present in the upper deep cervical nodes. Coagulation diathermy in such cases as described is fraught with danger because of the proximity of the internal carotoid artery and jugular vein. Irradiation therapy has not been productive of many cures. Contemplation of this situation encouraged Raven to perform a radical operation which conforms to the surgical principles of the treatment of cancer that the primary disease must be widely excised together with the lymphatic vessels and regional lymph nodes, if possible, in continuity. Raven describes the operative procedure briefly. An incision is made on the side of the lesion starting at the tip of the mastoid process and curves forward one inch below the angle of the mandible to the midline one inch below the symphysis menti. It then passes down the midline of the neck to one-half inch above the suprasternal notch and outwards above the clavicle to the posterior border of the sternocleidomastoid muscle. Block dissection of the neck is carried out, and the usual structures are removed. The buccal stage of the operation is carried out through the open mouth, the involved structures being excised with the diathermy knife. These structures are delivered into the cervical wound and excised completely, and the cervical operation is continued to its conclusion. The patient on whom this operation was performed has survived a three year period. Because of the unique approach and its originality, this article is considered excellent. Patients with such lesions as described by Raven are too often referred to the radiologist with the full knowledge that irradiation offers

little or no hope of survival. Lateral pharyngotomy offers some hope of success in such cases. This new approach is worthy of much consideration.

Cancer of the hypopharynx is practically undetectable in its early stages; however, as it progresses, it usually interferes with respiration and deglutition, producing much suffering, and it is attended with a high mortality. Thus, as Raven[53] points out, "any form of treatment that will relieve the patient's suffering, and is also designed to effect a cure, is welcomed." Contemplation of the serious plight of these patients stimulated Raven to study the surgical problem involved, which resolved itself into radical extirpation of the hypopharynx plus any metastatic lesions. He presents his experience in the radical surgical treatment of 18 patients with carcinoma of the hypopharynx. Most of the patients had an advanced stage of the disease manifested by dyspnea, dysphagia and stridor; some had received radiotherapy, but the disease continued to progress. There was no operative mortality. Ten patients were alive at the time of Raven's report, from two to 34 months postoperatively; six died of cancer from four to 23 months postoperatively and two died of other causes two and 10 months after operation.

Raven advocates laryngo-pharyngectomy and laryngoesophago-pharyngectomy for more extensive cases. These are well described and in cases permitting, the one-stage procedure is used. In more extensive cases the two-stage procedure is employed, the patient being left with a temporary pharyngeal fistula. In four to six weeks plastic repair with closure is accomplished. Carcinoma of the hypopharynx carries a high mortality rate, and to date radiation has offered little or no relief. The results obtained with radical extirpation justify more attempts in effecting cures in these cases.

According to Bullock and Snyder[54], invasive carcinoma of a pharyngeal diverticulum has been reported on several occasions; however, review of the literature revealed no instance of non-invasive carcinoma in a similar site. Hence, they present a case of carcinoma *in situ* in the pharyngeal diverticulum of a man 66 years old. The usual symptoms diagnostic

of a pharyngeal diverticulum were present and by a one-stage operative procedure the diverticulum was excised and the defect repaired. Nothing unusual was noted in the removed sac, but on microscopic examination a report of carcinoma *in situ* was rendered.

In a scanty report, Daito and associates[55] present an analysis of 68 cases of malignancy of the nasopharynx reported in the Japanese literature since 1941. There were about equal numbers of sarcomas and carcinomas. Twenty-eight per cent of the carcinomas and 22 per cent of the sarcomas showed cranial nerve involvement when first seen. Treatment consisted in surgical removal after first splitting the palate followed by radium implantation or Roentgen-ray therapy. Results were poor, cure being reported in only 10 patients.

In an interesting article on malignancies of the upper air passages Kearny[56] states that too much emphasis cannot be laid on the necessity of early diagnosis. His discussion of the method of treating malignancies of the paranasal sinuses is well presented. He summarizes this excellent article by saying that radium and Roentgen therapy play an important part in the treatment of malignant growths of the nose, paranasal sinuses and nasopharynx. Surgical removal of malignant tumors of the paranasal sinuses with or without postoperative radiation is the method of choice in selected cases. He correctly says that best results occur when treatment is instituted early, while the growth is accessible, limited in extent, has not metastasized and has not invaded cartilage or bone. Malignant lesions of the nasopharynx are best treated by irradiation. Kearny thinks the choice of surgery, radiotherapy or both in cases of malignant disease of the nose, paranasal sinuses or nasopharynx can best be determined by a team consisting of a rhinologist, pathologist and a radio-therapist. Radium and Roentgen therapy play an important part in the treatment of malignant lesions of the tongue, tonsils and hypopharynx. Surgery is advised in metastatic lymph node involvement from lingual carcinoma and block dissection of the cervical lymph nodes and should

be performed at the time the lesion is removed from the tongue. It is thought that best results in malignancies of the tonsils are obtained by radio-therapy. Malignancies of the hypopharynx are discussed and much can be offered these patients.

Laryngologists interested in malignancies should read this article thoroughly. We would like to include, in this review, the entire article as presented by Kearny on the treatment of malignancies of the upper passages, for it is replete with good worthwhile suggestions.

A good analysis of the neurologic symptoms produced by nasopharyngeal tumors is presented by deOya and Segovia[57]. A splendid discussion is given of the various syndromes produced by these lesions. Twenty-eight cases of malignancies of the nasopharynx are carefully analyzed; of these 22 patients presented neurologic symptoms. All but two of the 28 patients had evidences of cervical metastasis and in only five was the metastasis unilateral. The initial symptoms presented were those of advanced nasopharyngeal lesions, and the high percentage of cervical metastasis forces us to the conclusion that these cases were seen in consultation when neurologic symptoms developed or else the patients reported for medical care extremely late.

Geist and Portmann[58] present a study of 72 consecutive cases of primary malignant tumors of the nasopharynx encountered between 1935 and 1949. Malignant tumors of the nasopharynx comprise from 1 to 2 per cent of the total number of cancers. It is most interesting that such a high incidence of primary nasopharyngeal malignancy is found in Chinese. During an eight-year period at the University of Hong Kong, 419 cases of malignancies were seen of which 114 were carcinomas of the nasopharynx. Such malignancies are more common in men than women. Geist and Portmann divide these tumors into three pathologic types: ulcerated, lobular and exophytic, each of which is described in detail. Unfortunately, the signs and symptoms of nasopharyngeal cancer are seldom recognized by the medical profession. No one sign or symptom is pathognomonic of this type of malig-

nancy. Keen interpretation of the presenting symptoms will lead to a proper and careful examination and a correct diagnosis. Symptoms and diagnosis are discussed at length and afford interesting reading. Radiotherapy is accepted almost universally as the only treatment for malignant tumors of the nasopharynx. The technique of radiation therapy is described. Frequently an attempt is made to irradiate the lateral fields of the neck. It is generally agreed that the prognosis is poor because of the usually late diagnosis; however, life expectancy in the treated patient is more than twice that of the untreated one.

Goldmann and Engbring[59] present a case of extrahepatic biliary obstruction due to metastases from a locally "cured" nasopharyngeal malignant growth which had been treated one year previously by combined excision and irradiation.

In an excellent article which bears reading in its entirety Dunlap[60] discusses the problem of treatment of carcinoma of the laryngopharynx and cervical esophagus and presents a historical review. Various misconceptions are clarified; such as extirpative surgery of this area is not attended by a high mortality and radio-therapy is of little or no value in the treatment of carcinoma of this area.

Surgical techniques based on major excision in 20 cases are discussed in detail and well illustrated. Postcricoid carcinoma or extensive laryngeal carcinoma which have invaded the pharynx require pharyngolaryngectomy with subsequent pharyngo-esophageal reconstruction in stages. Experience in one case suggests the possibility of primary reconstruction of the pharynx by the use of a split thickness skin graft covering a specially molded polythene tube.

Within a period of 18 months Chene and associates[61] saw eight patients in whom cancer developed in the esophagus some time after they had been treated for oropharyngeal carcinoma and there was clinical evidence of cicatrization of the oropharyngeal region. These cases are briefly described. Chene and associates believe that such an occurrence is not nearly so rare as reports in the literature would indi-

cate. They do not believe that these oro-pharyngo-esophageal epitheliomas represent recurrences, but rather they consider them as successive manifestations of a mucosa of the same embryologic origin or histologic type of a truly generalized cancer whose localization is multiple or repeated.

Piquet and associates[62] made a study of 132 cases of carcinoma of the hypopharynx treated by radiation and surgery since 1944, 118 of which were followed long enough to evaluate the results. From their study they concluded that radiation alone had little effect on malignant epithelial tumors of the hypopharynx and cures were exceptional in the patients they treated. Surgical treatment alone was likewise not very effective. Their best results were obtained in those patients treated by combined radiation and surgical therapy. Particularly resistant to treatment were tumors located in the posterior and lateral walls of the hypopharynx. Their study indicated that extensive surgical procedures were less dangerous in their immediate results and more favorable in their remote results than limited procedures. They consider particularly aggravating adenopathy which is recognized preoperatively or even at operation; however, they point out that if it develops postoperatively in spite of radiotherapy, adenectomy will give successful final results.

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NINETEETH INTERNATIONAL CONGRESS OF OTO-NEURO-OPHTHALMOLOGY.

The Nineteenth International Congress of Oto-Neuro-Ophthalmology will be held on the occasion of the commemoration of the foundation of São Paulo, from June 11 to 17, 1954, in the city of São Paulo (Brazil).

The official themes are:

1. Metabolical and avitaminotic disturbances in oto-neuro-ophthalmology.
2. The physiopathology of the facial nerve.

These themes are subdivided into various topics, and official speakers from different countries have been invited to prepare special themes.

The official languages are: Portuguese, Spanish, English and French.

All physicians, specialists in oto-rhino-laryngology, neurology and ophthalmology may apply for membership.

Prof. Cyro de Rezende is President of the Congress, and Dr. Luiz Piza Neto is general secretary.

Inquiries or suggestions about the Congress should be addressed to: Clínica Oftalmológica, Hospital das Clínicas, São Paulo, Brazil.

ELECTRON MICROSCOPIC AND X-RAY DIFFRACTION STUDIES OF STATOCONIA.*

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There are somewhat conflicting views expressed in the literature regarding the exact structure and composition of the crystalline bodies in the static organ of vertebrates.

In higher mammals the statoconia (also called otoconia) are embedded in a jellylike mass on the maculae of the utricle and the saccule. Similar formations can also be found in the labyrinth of lower vertebrates, but in most fishes they form large, irregular statoliths containing layers of calcium carbonate.

Calcium carbonate can crystallize in three different modifications: calcite, aragonite and vaterite. Calcite is the stable modification and a very common mineral; Aragonite is rather rare, and can easily be transformed to calcite by heating, even at normal temperature it changes very slowly to calcite: Vaterite does not occur naturally.

Funaoka and Toyota[1] (1928) demonstrated by means of X-ray diffraction technique that the statoconia in frogs consisted of aragonite. Hastings[2] investigated statoconia from a salamander (Amblystoma tigrinum), and found that they were composed of 74 per cent aragonite and 26 per cent dhallite (carbonate hydroxyapatite). The formation of statoliths in shark (Squalus acanthias) has recently been discussed by Vilstrup.[3]

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In man, statoconia are generally described as small calcium carbonate crystals forming rather regular hexagonal prisms with pointed ends. Kolmer[4] found their approximate size to be 3 by 5 μ.

Their specific weight has been given as 2.93—2.95 by Polyak, McHugh and Judd.[5] The nature of their crystallographic structure has been described by Brandenberger and Schintz[6] who used the powder X-ray diffraction technique. They found that the diffraction pattern coincided with that of calcite. They considered each statoconium to be a single crystal. The specific gravity of calcite is 2.71 and that of aragonite 2.93; thus there is a discrepancy between the X-ray finding and the specific gravity. The latter corresponds to aragonite as found in fish statoliths.

This paper reports the results obtained when studying statoconia in man, rabbit and guinea-pig by means of electron microscopy and X-ray crystallography.

ELECTRON MICROSCOPY.

After localizing the maculae, a relatively great number of statoconia could be scraped off. In polarized light these statoconia appeared as elongated particles showing approximately parallel extinction and behaving like single crystals.

In the electron microscope, human statoconia appeared as particles of approximately uniform shape but of varying sizes (see Fig. 1). The lengths of statoconia varied from 1 to 20 μ and the diameter from less than 1 to about 8 μ. They generally had pointed ends, and often slightly curved sides as illustrated in Fig. 1. Some statoconia, however, had somewhat rounded edges, but this seemed to depend upon the angle from which they were observed (probably more or less end on view). The surface of the long sides of the statoconia were found to be smooth, but the ends were uneven and serrated in certain projections (see Fig. 5).

When the statoconia were crushed and the fragments examined under the electron microscope, numerous thin, hexagonal platelets were found (see Fig. 6). These hexagonal

fragments had 120° angles, and the statoconia were apparently built up from close packed layers of such hexagonal platelets.

In the central part of the statoconia dustlike "nuclei" are

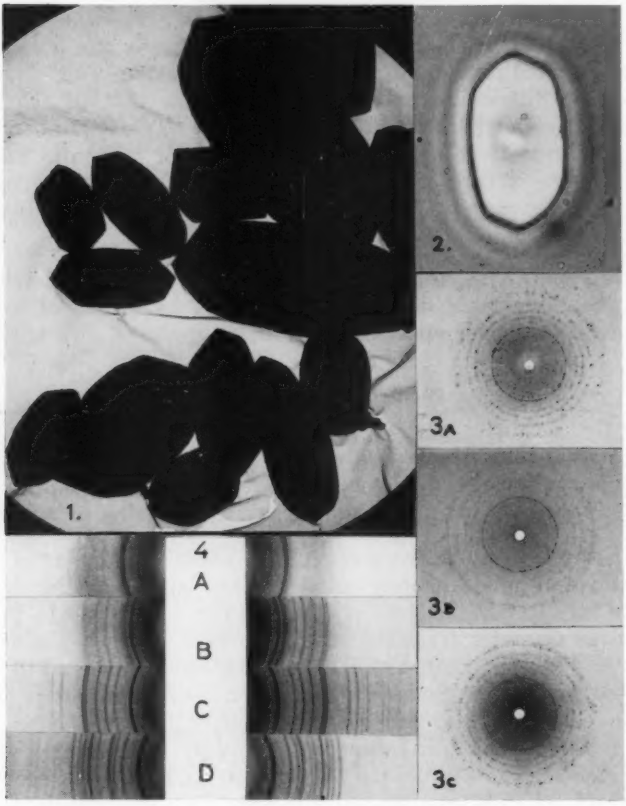

Fig. 1. Statoconia of different sizes from man. Electron micrograph. 10,000 x.

Fig. 2. Statoconium from man, phase contrast. 5,000 x.

Fig. 3. Micro diffraction pattern of statoconia from rabbit (A) compared with the pattern of guinea-pig (B) and calcite (C).

Fig. 4. X-ray diffraction patterns from (A) bone tissue, (B) human statoconia, (C) calcite, (D) aragonite.

often found. These structures can be stained with hematoxylin and have been described by Schwalbe (1892), and Herzog,[7] (1925). They can also be seen in the phase contrast microscope (see Fig. 2). Attempts to study these nuclei in the thin sections of decalcified statoconia were not successful.

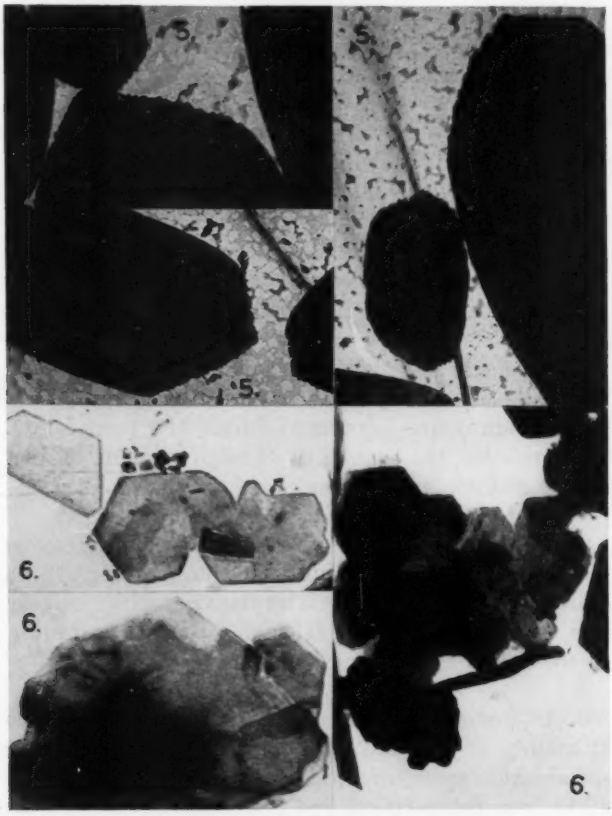

Fig. 5. Electron micrographs of statoconia from man, showing rather even long sides and serrated ends. Enlargement around 25,000 x.

Fig. 6. Fragments of crushed statoconia from man, which have broken up into a number of thin plates. The angles of the plates are always 120°. Enlargement 50,000 x.

X-RAY DIFFRACTION.

Only small amounts of statoconia were available; therefore, the micro X-ray diffraction camera described by Chesley[8] was used. The X-radiation was nickel filtered CuKα. Fig. 3 shows diffraction patterns of statoconia from rabbit (A) and guinea-pig (B) and the pattern of calcium carbonate in the form of calcite (C). As can be seen, they have identical interplanar spacings. The particle size in the diagrams of the statoconia is of the order of 10^{-3} cm.

Statoconia from man were examined in an ordinary Deby-Scherrer camera with a cylindrical film (diameter of the camera 57.4 mm). Nickel-filtered CuKα-radiation ($\lambda = 1.54$A) was used. The patterns shown in Fig. 4A—D come from bone tissue, statoconia, calcite and aragonite. It is clearly demonstrated that the statoconia (see Fig. 4B) have a calcite pattern (see 4C) and this was further confirmed by precision measurements with monochromatic CuKα-radiation in a Guinier camera. In addition, lines indicating the presence of bone salt (hydroxyapatite) can be seen. This bone salt may be an admixture introduced during the preparation of the statoconia but the possibility of small amounts of apatite within the statoconia cannot be completely ruled out.

It is clearly demonstrated that there is no resemblance between the pattern of human statoconia (see Fig. 4B) and aragonite (see Fig. 4D). The interplanar spacings calculated for the third polymorph of calcium carbonate, vaterite, do not fit the values found for statoconia.

Thus statoconia from man, rabbit and guinea-pig are composed mainly of calcium carbonate in the form of calcite (rhombohedral, space group D^6_{3d}). Diffraction patterns of statoliths from fishes (perch and sole), however, gave aragonite patterns (orthorhombic, space group V^{16}_h). Rotation photographs of single statoconia will be obtained later when micro-beam X-ray rotation techniques become available.

SUMMARY.

Statoconia from man have been investigated by electron microscopy and X-ray diffraction. They consisted of calcite as with statoconia from rabbit and guinea-pig. Fish statoliths, however, were composed of aragonite.

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TUBERCULOUS OTITIS MEDIA.*

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Tuberculosis of the ear has been described extensively for many years, beginning with the early European otologists and continuing through to modern times. The advent of the antimicrobial drugs effective against tuberculosis has ushered in a new era in the treatment of this disease. The impact has been especially great on the extrapulmonary mucosal lesions, such as the larynx and bowel. A study seems opportune to determine if the prognosis and treatment of tuberculous otitis media has been similarly improved. This paper depicts observations of various aspects of tuberculosis of the ear, including the results of treatment with the antimicrobial drugs.

Wilde,[1] in his textbook of 1853, wrote that "tuberculous deposits have been found in the ear, and also in connection with the auditory nerve." Von Troltsch,[2] in 1869, stated that tuberculosis of the temporal bone was very rare. He described so-called tuberculous deposits as probably being due to masses of shrunken pus. He thought miliary and pulmonary tuberculosis could be caused by these primary foci in the ear. Bezold[3] and Politzer[7] gave accurate descriptions of the disease, including the histopathology and bacteriology.

The contributions on this subject have been so numerous over the years that space prevents review or even mention of all of them. Authoritative descriptions have been presented by Spencer,[8,18] Kerrison,[6] Meyerson,[14,17] Lederer,[20] Miller,[13] Cox and Dwyer,[10] Fowler,[19] St. Clair Thomson,[12] Ballenger,[16] Ormerod,[11] Leegard,[5] Lussman and Bendove.[9] Detailed histopathologic studies were carried out by Turner and Fraser[4] and Proctor and Lindsay.[15] From these articles there has

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emerged the concept of a characteristic type of chronic otitis media, the salient features of which are the painless onset of aural discharge, multiple perforations which soon coalesce into one rapidly enlarging opening, exuberant granulations, early and severe hearing loss, necrosis of the ossicles and of areas in the temporal bone, with the frequent onset of facial paralysis and fistulae of the labyrinth.

PRESENT STUDY.

The patients in this investigation were seen in consultation during the past five years at two large tuberculosis hospitals. This has afforded the opportunity for observation of a comparatively large number of patients with otitis media. There were certain limitations in the studies, follow-up and treatment, but whenever feasible the following investigations were carried out: A history of the time and mode of onset of ear symptoms, otoscopic examination, hearing tests, including the whisper voice, tuning forks and audiometer, X-rays of the mastoids, culture of the discharge and observation of the response to antimicrobial therapy. Death, transfer or discharge, the severity of the pulmonary disease, and surgery prevented some of the contemplated studies on many of the patients.

ETIOLOGY.

From the many patients observed with otitis media, 52 were considered to have presumptive evidence of tuberculosis of the ear. (The criteria which determined this diagnosis will be discussed later.) This number would indicate that it is not a frequent complication of pulmonary tuberculosis. For purposes of comparison, over 200 patients in the same hospital population had the diagnosis of tuberculous laryngitis. Involvement of the ear is the second most common otolaryngologic disease of tuberculous etiology. The patients seen at these two hospitals were mostly adult males and were all being treated for pulmonary tuberculosis.

Specific infection of the ear is considered to be either primary or secondary to a focus elsewhere, most commonly the

lungs. Primary aural tuberculosis is presumed to occur usually in infants and children, although Proctor and Lindsay[15] doubted that the disease in the ear is ever primary. Their explanation is that the ear becomes involved by the bacillemia which occurs with the hilar gland infection from the Ghon tubercle (Ranke's complex, primary complex). The lung lesion is not demonstrable later, when the ear disease becomes manifest.

Secondary infection of the middle ear may be either hematogenous or by way of the Eustachian tube. Various writers regard one or the other method as the usual path of infection. Politzer,[7] Cox and Dwyer,[10] Kerrison,[6] Miller,[13] Turner and Fraser,[4] Ormerod[11] and Thomson[12] thought that the tube was the common route. Certainly the constant cough with bacilli laden sputum of pulmonary patients makes this seem likely. Meyerson[14,17] and Proctor and Lindsay[15] believe the infection occurs from the blood stream. There was no evidence obtained in the present study which would tend to indicate by which avenue the ear became involved.

ONSET AND COURSE.

The most frequent symptom was the appearance of aural discharge. Rarely was there any pain; only two patients mentioned a slight discomfort at the onset of discharge. Other frequent complaints were tinnitus, a feeling of fullness or blocking of the ear, and defective hearing. The latter symptoms were sometimes present weeks or even months before the occurrence of discharge. Several patients were seen because of tinnitus and poor hearing in one ear. The drum membranes were normal, the Eustachian tubes were patent, and there was no improvement on inflation. The functional tests indicated a conduction type of hearing impairment. The diagnosis of unilateral otosclerosis seemed the most plausible one. The subsequent appearance of inflammatory alterations of the drum membrane, or of discharge, revealed the true nature of the disease.

Comment: This observation of the painless, insidious onset of aural discharge was made by all writers. It is one of the most characteristic features of the disease, and most important in the diagnosis.

OTOSCOPIC FINDINGS.

The description of the drum membrane in tuberculous otitis media may be divided into two stages, the alterations seen before and after perforation. Eleven patients (see Figs. 1,

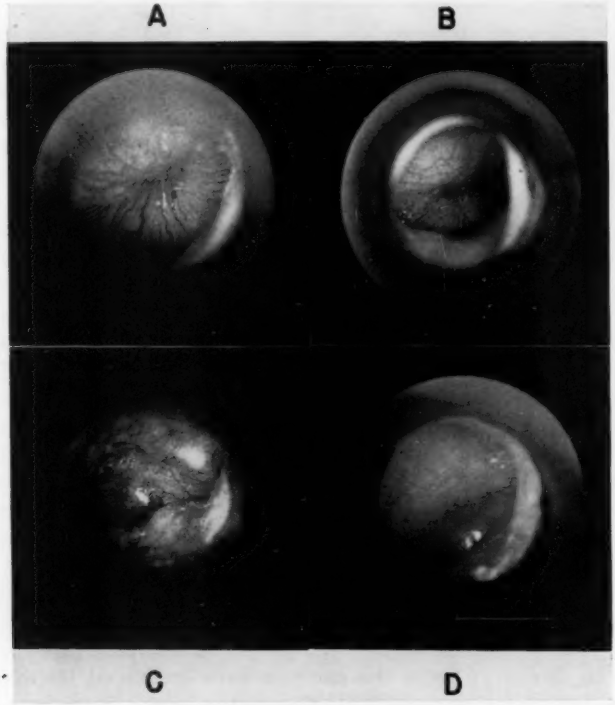

Fig. 1. Copies of kodachromes showing examples of the appearance of the drum membrane at the onset of tuberculous otitis media, before perforation has occurred. All reveal thickening and hyperemia; A, B and D also reveal bulging.

5A, 6A) were seen before rupture of the drum had occurred. The complaints which called attention to the ear were tinnitus, a feeling of fullness, or defective hearing. The drum was seen to be greatly thickened, intensely hyperemic, the landmarks were obliterated, and in some instances there was bulging. The appearance was similar to that seen in the usual acute nonspecific otitis media. The vessels of the drum (see Figs. 1, A, B, C, 5A, 6A) were often greatly enlarged and engorged, as described by Miller. There was no pain or febrile reaction. This same appearance would be seen to last weeks or longer before perforation would occur.

A yellowish spot would appear which might persist some time, until finally a perforation would become manifest, with escape of thin purulent discharge. In a few instances there were multiple small perforations which soon coalesced and enlarged rapidly. As a result the opening in the tympanic membrane shortly appeared like that seen in a nonspecific discharging ear of long standing. The perforations first observed after the ear had been discharging for some time were rarely multiple, this being noticed only four times. The perforations were of all sizes and shapes (see Figs. 2, 3, 4, 8), with little to distinguish them from nonspecific chronic ears. The size was not unusual in most of the patients, but in five individuals it was seen to be practically complete (see Figs. 2, A, D, 8B). In two instances the manubrium was seen to be bared of its mucosal covering, so that it had the appearance of cadaveric bone (see Fig. 2A). One patient had a large area of bare bone in the external canal, with a sequestrum from the region of the annulus. Exuberant granulations were not common, being found in only four instances. Three patients had multiple draining sinuses, either about the ear or from the external canal, with periotitic swelling. This is the childhood form of the disease, described by many authors.

The discharge from the ear was usually thin at its inception, later becoming more mucoid and thicker. There was no odor and the color was not distinctive. The amount varied, but usually was not copious.

Comment: This occurrence of acute reaction of the drum membrane without pain has been described by most writers. When seen in an individual with pulmonary tuberculosis, it is practically diagnostic of specific involvement of the ear.

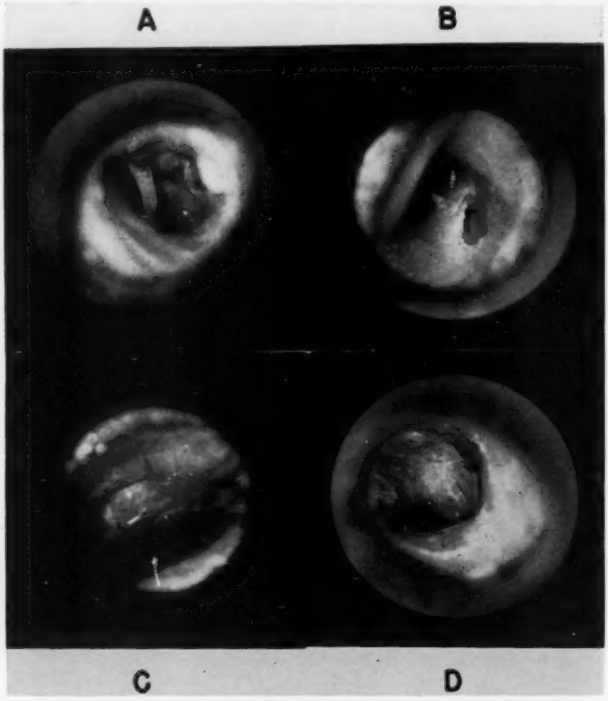

Fig. 2. Appearance of the perforations seen in tuberculous otitis media. (A) Left ear has been discharging for seven months. The manubrium is bared of its mucosa, giving it a cadaveric appearance. (B) Right ear has been discharging for 18 months. (C) Recent perforation of the right drum membrane revealing pale granulations in the middle ear. (D) Left ear has discharged for three years. Total destruction of the drum membrane with apparent extrusion of the ossicles.

HEARING STATUS.

Defective hearing was a common early symptom. As previously mentioned, in several instances the hearing defect

preceded inflammation of the drum membrane. The hearing loss was usually severe from the onset, more so than would be expected from the duration of symptoms or the appearance of the drum membrane. Hearing for the whisper voice was reduced to one foot or less. The audiogram revealed an average 40 to 50 db loss (see Figs. 3, 4). This would be apparent early, but did not seem to progress much below this level, even in those who had had discharge for some time. Thus

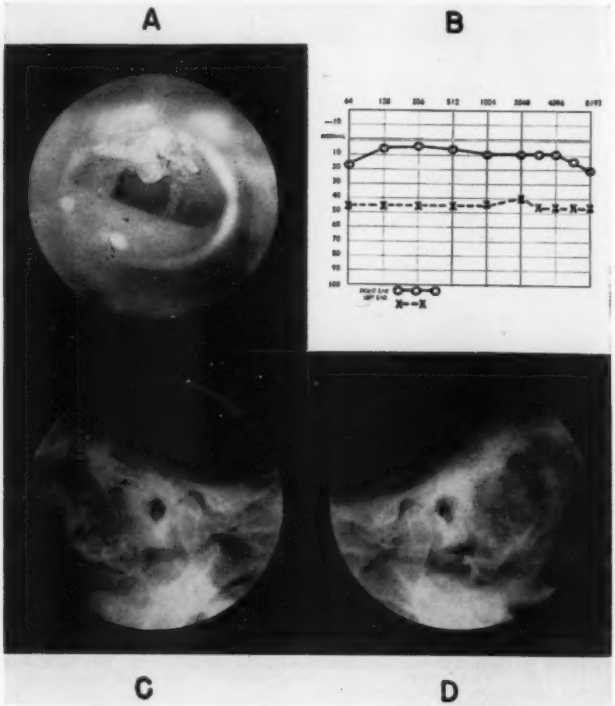

Fig. 3. (A) Appearance of the left drum membrane of a 50-year-old male with discharge from the ear for over two years. The ears had appeared normal when seen previously for tuberculosis of the larynx. (B) Audiogram, made after the ear had been discharging for two years. (C) The right and (D) the left mastoid X-ray. Both are well pneumatized; the left has increased density.

the hearing loss was severe enough in the early stages to be of diagnostic significance. This was not apparent in disease of long standing where the impairment resembled that seen in nonspecific discharging ears.

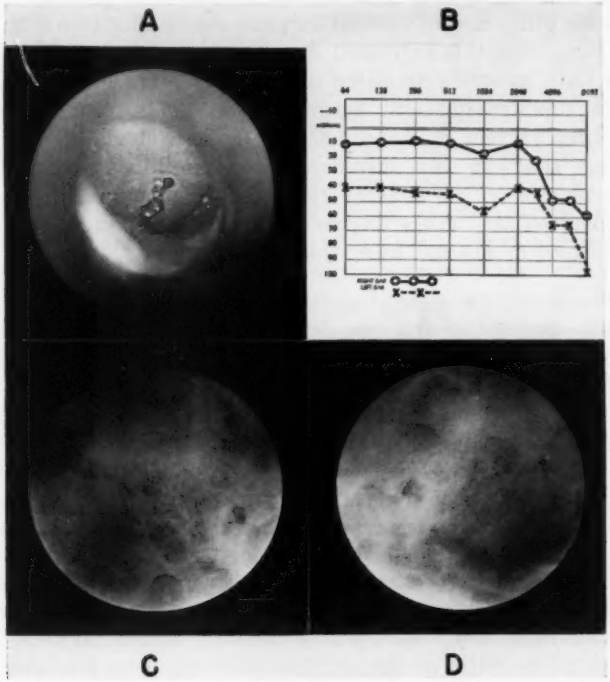

Fig. 4. (A) Appearance of the left drum membrane of a 38-year-old male with painless onset of discharge two months before. The ears were seen to be normal when he was examined for tuberculous laryngitis three months before. Culture negative for M. tuberculosis. (B) Audiogram of same patient. (C) The right and (D) the left mastoid X-ray. Both are well penumatized, normal.

The conduction character of the loss was maintained, with the Weber lateralized to the affected ear, and negative Rinne test. This differs from Politzer's findings of decreased bone conduction and an inner ear type of loss which he attributed to involvement of the labyrinth.

X-RAY STUDIES.

Roentgenograms were made of 36 of the patients in this series. These usually revealed bilateral pneumatization of the mastoids, with increased density of the cells of the affected ear. The appearance was similar to that seen in acute nonspecific otitis media, without surgical mastoiditis (see Fig. 3). In some even this increased density of the cells was lacking, and both mastoids appeared quite normal (see Fig. 4). Two films revealed bilateral apneumatic mastoids, four revealed varying degrees of apneumatization or sclerosis on the affected side. In two instances the mastoid was completely apneumatic on the affected side, with pneumatization of the uninvolved ear. There was no evidence of softening of the cell partitions or abscess formation.

Comment: In chronic otitis media, the usual finding is so-called sclerosis, or the apneumatic, infantile type of mastoid. It is most unusual to find extensive pneumatization. The Wittmaack theory postulates that the dense bone is not the result of infection, but is due to a lack of development of the cells. This also explains the other "infantile" characteristics of low lying dura and forward placed sigmoid sinus. The common finding of extensive pneumatization in tuberculous otitis media was interpreted as establishing the recent onset of the infection, and distinguishing it from the usual nonspecific chronic discharging ear. This distinctive X-ray finding was not mentioned in any of the articles reviewed. Bezold noted that there was a lack of "eburnized bone" in tuberculous otitis media, as differentiated from the nonspecific form. He thought that it indicated a lack of response to the infection with the M. tuberculosis. The Wittmaack theory would seem a better explanation.

BACTERIOLOGIC STUDIES.

A culture was made of the ear discharge in 31 of these patients. A platinum loop was used, small enough to be inserted through the perforation when the discharge was scant. The material was placed directly on Petragnani's media and

incubated. M. tuberculosis was demonstrated in only eight patients. Overgrowth of the media by other organisms was the common occurrence. This media requires incubation of from four to six weeks. When the culture was negative, further specimens were not made, since by then the patient had usually received considerable antimicrobial therapy as well as local medication.

This simple method of culturing the discharge was found to be inadequate and a method of first getting rid of other organisms, as described by Cox and Dwyer,[10] might well have given a higher percentage of positive findings. Politzer[7] and Bezold[3] told of the paucity of bacilli in the aural secretion and of the difficulty in demonstrating them. This is concurred in by most writers, all giving a comparatively low percentage of positive bacteriologic evidence in suspected ears. Myerson[14,17] mentioned that 5 to 35 per cent of suspected cases are positive. He was able to demonstrate the organism in 35 per cent of his cases by either smear, culture or animal inoculation. It has been shown that the use of streptomycin renders culture of the organisms more difficult. Many of the patients in this series had had antimicrobial therapy before the culture was made. This may have been a factor in the difficulties in demonstrating the presence of the bacilli in the aural secretion.

There was no correlation with the type of ear which yielded a positive culture. Ears observed from before perforation occurred gave negative culture results as often as those seen later in the course of the disease.

COMPLICATIONS.

None of the patients in this series manifested a facial paralysis while under observation. The Siegle speculum was used to test for the presence of labyrinthine fistula. No positive tests were elicited. One patient had sequestration of bone from the external canal. None of the other complications described as frequent sequelae of tuberculous otitis media

were found. There were no symptoms to suggest extradural abscess, petrositis or meningitis in any of the patients while under observation.

Most writers describe the frequent occurrence of these serious complications. On first thought it would seem unusual to study a large group of patients without finding any evidence of extensive destruction of the temporal bone; however, this agrees with the experience of Sir St. Clair Thomson,[12] and Miller,[13] who also reported a lack of complications in their patients. There are several possible explanations for this change in the severity of the disease, as compared to earlier times. One is that many aspects of tuberculosis are different today, in this country, due to improved nutrition and better general treatment of the disease. Another is that the patients in this series, as well as Miller's and St. Clair Thomson's, were mostly adults. Tuberculous otitis media seems to be quite a different disease in children. Many of the patients with serious complications described in the literature were infants and children. Most of Proctor's and Lindsay's, and Fraser's and Turner's patients were infants. The latter authors reported a 45 per cent incidence of facial paralysis in 60 cases. A third factor in the present series is antimicrobial therapy. All of these patients received streptomycin or para-aminosalicylic acid, or both.

DIAGNOSIS.

At the beginning of my work in tuberculosis, a phthisiologist remarked that any case of discharging ear developing in an individual known to have pulmonary tuberculosis could be considered to be specific in origin. This seemed a rash statement at the time, but further experience has confirmed its accuracy. Myerson[14] holds a similar opinion, "Our experience demonstrates that a discharge from the middle ear appearing without pain in a tuberculous individual should be considered as tuberculous." Thus the time and mode of onset becomes most important in the diagnosis. Additional items in establishing the diagnosis are:

1. The presence of a thickened hyperemic bulging drum

membrane in the absence of pain, or a rapidly enlarging perforation with a painless onset. Multiple small perforations that soon coalesce, and baring of the ossicles of their mucosal covering are evidence of specific involvement of the ear. *2.* Severe early hearing loss, out of proportion to the length of time the ear has discharged, or the appearance of the drum membrane. *3.* The X-ray findings of a well pneumatized mastoid in a chronic discharging ear. This finding helps determine the time of onset of the discharge. *4.* The definitive diagnosis is established either by demonstrating the Koch bacillus in the discharge, or by biopsy of granulations or operative specimens. Some or all of these were used in establishing the presumptive clinical diagnosis, or the definitive diagnosis in the fifty-two patients in this series. The presence of acid-fast bacilli was demonstrated in only eight patients. Eleven persons were seen with the characteristic drum membrane findings before perforation. In seven others the perforation was typical enough to justify a clinical diagnosis. This leaves a larger group in which the main evidence for diagnosing tuberculous otitis media was the painless onset of aural discharge in the presence of pulmonary tuberculosis. The following is offered as evidence that this is sufficient to make this presumptive diagnosis:

1. A study of the literature reveals all authors to be of this opinion.

2. A comparison with discharging ears where M. tuberculosis have been demonstrated reveals their similar clinical course.

3. The organisms are seldom isolated from the secretion of a number of extrapulmonary complications that are assumed to be tuberculous if the primary disease is proven, *i.e.*, pleural fluid, pericardial fluid, spinal fluid.

4. The clinical diagnosis of tuberculous laryngitis based on the history and gross appearance is commonly accepted without histologic proof.

TREATMENT.

The antimicrobial drugs used in the treatment of these patients were streptomycin, or its derivative, dihydrostreptomycin, and para-aminosalicylic acid (PAS). A number of different dosage schedules were used. Streptomycin was administered in doses of from 2 to 0.5 gm. daily, for from 60 to 120 days and longer. At present it is usually used with PAS, so-called combined therapy. Twelve gm. of PAS are given daily and 1 gm. of streptomycin twice a week, this schedule being kept up for many months. The use of PAS delays the development of organism resistance to streptomycin, allowing effective use of the drugs for prolonged periods. The toxic effects of streptomycin on the VIIIth nerve have received wide attention and will not be reviewed here.

As stated before, most of these patients were given chemotherapy for their primary disease, and treatment did not coincide with the onset of the ear complication. Due to organism resistance, a second course of streptomycin was likely to be less effective. There have been a number of new drugs which show real promise in the treatment of tuberculosis. One of the great benefits of having a number of drugs available is that when resistance has developed to one, another may be used to control the development of a complication. The isonicotinic acid derivatives were not available for use on any of the discharging ears in this series. They have been used with encouraging results in tuberculous laryngitis, and appear to be a real addition to the available agents for the treatment of tuberculosis.

The criteria for the evaluation of the results of treatment must be defined. Cessation of discharge from a chronic ear cannot be considered a cure since it leaves a large perforation subject to reinfection from the Eustachian tube or the external canal; furthermore, treatment may be of benefit even though the ear continues to discharge. This would be evidenced by the disease assuming a more benign course than might be expected without therapy.

Because the chemotherapy was given to these patients primarily for their chest disease, it is difficult to record the effects on the ear complication in tabular form. The results of therapy will be reported on a number of individual patients where the action could be evaluated accurately, and by the general impressions gathered from observation of the entire group.

Two patients were seen before perforation, with the thickened hyperemic drum membrane previously described (see Figs. 5 and 6). They were given chemotherapy, and neither

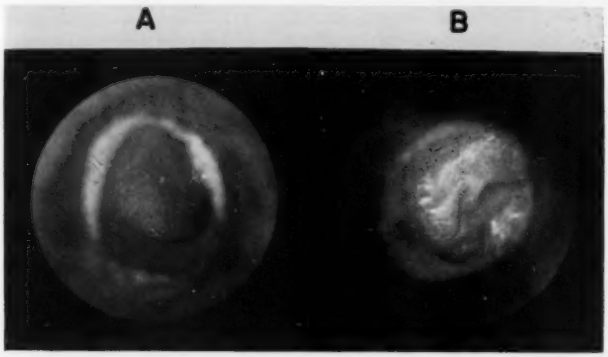

Fig. 5. (A) Appearance of the drum membrane in a patient complaining of tinnitus, blocking and poor hearing for four weeks. The membrane is thickened, hyperemic and bulging. (B) Appearance of the drum membrane after one month of combined therapy, dihydrostreptomycin and PAS. Perforation did not occur, the membrane remains thickened, wrinkled without hyperemia.

progressed to perforation. In one (see Fig. 5B) the hyperemia disappeared, but the drum remained greatly thickened, with a pebbly appearance, like wrinkled leather. The hearing did not improve. In the second patient (see Fig. 6B) the alterations of the drum cleared up after treatment.

Two patients were seen before perforation but rupture was seen to occur in spite of starting treatment by chemotherapy; however, the discharge soon ceased and the perforation closed.

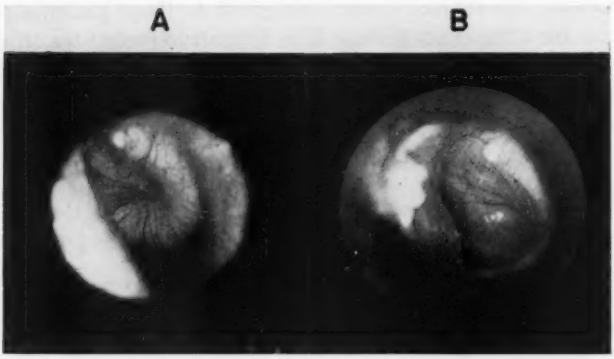

Fig. 6. (A) Appearance of the left drum membrane of a patient that complained of a "popping" in the ear. He has been deaf for many years and wears a hearing aid. The membrane is thickened, intensely injected with dilated vessels and bulging. (B) After five months of combined therapy. The drum membrane appears normal except for a few dilated vessels.

The drum membrane became thickened, scarred, retracted and adherent (see Fig. 7). There was no improvement in the hearing.

Three patients were seen with multiple draining sinuses in the external canal and about the ear, with periauricular swelling (the childhood form of the disease). After antimicrobial therapy the fistulae promptly closed, with cessation of the aural discharge and swelling.

One individual had a mastoidectomy some months before entering the hospital for tuberculosis. The external canal was filled with granulations with profuse discharge. A culture yielded M. tuberculosis. He was given streptomycin but no local therapy of any kind. The granulations promptly disappeared, and the ear became dry, revealing a very adequate radical cavity.

The majority of patients were seen after the ear had been discharging for some time. If they were not streptomycin-resistant, and this drug was administered, the usual result

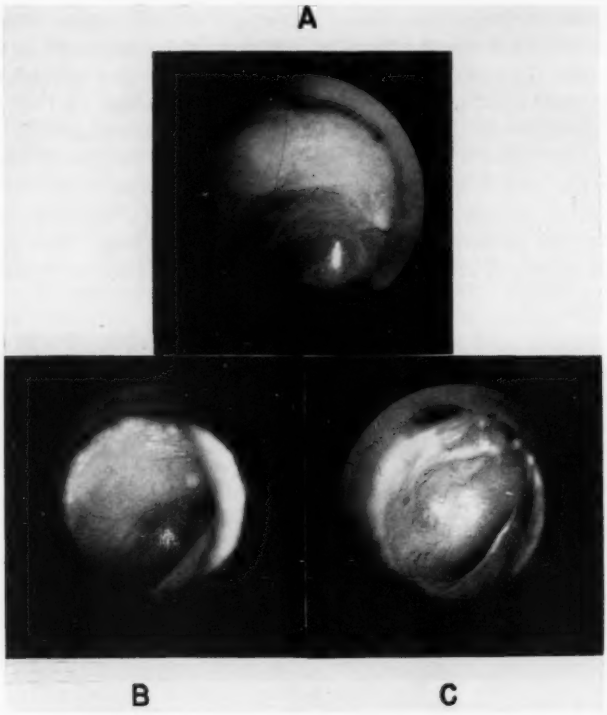

Fig. 7. (A) Appearance of the right drum membrane of a patient when seen for tuberculosis of the larynx. He was given a course of antimicrobial therapy. (B) appearance of the right drum membrane 11 months later. The right ear had begun to discharge one month before. The membrane is thickened, hyperemic and bulging, with a perforation in the yellow spot near the umbo. (C) Appearance seven months later, after local treatment with a 20 per cent solution of PAS in glycerine. The perforation has healed, the membrane is thickened and adherent to the promontory. There was no improvement in the hearing.

was prompt cessation of the discharge (see Fig. 8). The hearing remained poor. When seen for long periods, there would often be return of the discharge. This could have been due to reinfection by nontuberculous organisms.

Reference has been made to the lack of complications and to the comparatively benign course of the disease in all of the

patients in this series. Other observers[12,13] had similar experience, before a specific therapy was available; however, it was felt that the antimicrobial therapy these patients received was responsible for altering the course of the disease so that the ear became relatively innocuous, and not a menace to the patients' recovery from their tuberculosis. Lederer[20] states that the pathology of tuberculous otitis media is primarily an infection of the mucous membrane of the tympanum, with secondary involvement of the periosteum and bone. The beneficial effects of streptomycin in the mucosal lesions of tubercu-

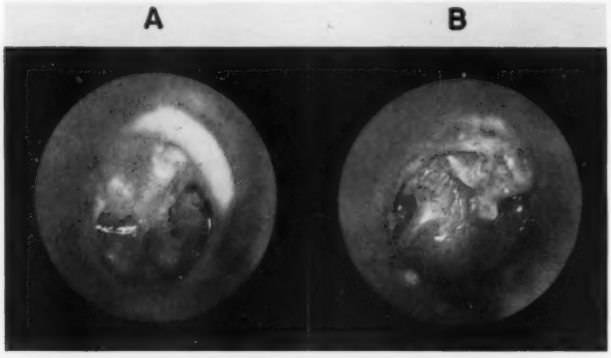

Fig. 8. (A) Appearance of the right ear in a 12-year-old Mexican girl with aural discharge for one year. There is a large perforation with exuberant granulations. (B) Same patient 10 months later after continuous combined therapy, as well as PAS in glycerine used in the ear. The granulations disappeared, the ear became dry and has remained so.

losis is well known. Most of the patients in this series received chemotherapy early in the course of the otitis media. It seems reasonable to assume there was amelioration of the mucosal disease, preventing progression to the periosteum and bone.

The main reliance was on systemic chemotherapy. In those where discharge persisted, local therapy was used. Streptomycin was not used in the ear locally, because of its known slight effect in tuberculosis unless given systemically. A 20 per cent solution of PAS in glycerine was used locally in five cases. In

three the ears became dry, and a specific local action was presumed. The usual solution of boroalcohol seemed to benefit some cases.

None of the patients in this series underwent mastoid surgery for the reason that a clear-cut indication was lacking. Many writers consider the results of surgery are of dubious value. Myerson[14] advised a radical mastoidectomy if any of the following complications develop: facial paralysis, subperiosteal abscess, labyrinthitis, mastoid tenderness and headache. He regards the absence of indications for operation in a large series of cases as pure coincidence. As stated before, the lack of complications in the patients in this series was presumed to be due, at least in part, to the fact that they had all received antimicrobial therapy. The three patients with the childhood form of the disease had prompt relief of all signs of the disease after treatment by chemotherapy.

CONCLUSIONS.

The following conclusions were reached as the result of this study:

1. The characteristic features of tuberculous otitis media described in the literature were, in general, confirmed. The benign clinical course and lack of complications was in contrast with most descriptions of this disease.

2. The diagnosis of specific infection of the ear depends largely upon the history of painless onset of aural discharge in an individual with pulmonary tuberculosis. The otoscopic appearance may not be unusual, and demonstration of acid-fast organisms may be most difficult.

3. The finding of a well pneumatized mastoid in chronic otitis media may be evidence of tuberculous etiology.

4. The treatment and prognosis of this disease have been completely altered by the advent of antimicrobial agents effective against the Koch bacillus. If seen ear-

ly, before perforation has occurred, chemotherapy may result in complete resolution of the tuberculous process in the ear.

5. All ear complaints in tuberculous individuals, especially tinnitus, a feeling of fullness, or loss of hearing should receive prompt attention. If the diagnosis can be made early, and chemotherapy administered, the onset of perforation may be prevented, with preservation of hearing.

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THE EFFICACY OF A WETTING AGENT (DUPONOL-C) AS AN AID IN THE TREATMENT OF LARYNGOTRACHEOBRONCHITIS.

PRELIMINARY REPORT.*

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For many years laryngotracheobronchitis has been a troublesome disease to treat. Gittins,[1] in 1936, stressed the fact that the mechanical obstruction was more serious than the type of infection. Many methods have been advocated to help in alleviating this dangerous complication.

In 1939, Evans[2] reported several cases in which he used a bacteriophage to liquefy the bronchial secretions with some apparent success. In the same year Galloway[3] suggested postural drainage as an aid in expelling the bronchial secretions. Davison,[4] in a paper read before the Eastern Section of the American Laryngological, Rhinological and Otological Society, Inc., in Pittsburgh in January, 1940, entitled "Some Observations on the Control of Temperature and Humidity in Oxygen Tents," emphasized for the first time the importance of humidity and temperature control in the handling of this obstructive problem. Orton[5] and many others[8] have since further emphasized the importance of this. Many solutions have been suggested as aids in removing the gummy bronchial secretions. Orton[5] recommended irrigation and aspiration with saline, sodium bicarbonate solution, or a 1 per cent solution of monochlorophenol. Cassidy[6] used a saline solution of ephedrine (1:1,000) to which a few drops of glycerin were added. Walsh[7] advocated the use of saline with neosynephrine or tuamine instilled through the tracheotomy tube, followed by suction aspiration.

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In 1950, Gorman[9] reported some cases in which a wetting agent (Duponol-C), used in a 0.1 per cent concentration, produced dramatic improvement in aspiration of the gummy secretions. He also reported that the addition of this agent to the water in a steam vaporizer caused the relative humidity to be raised from 65 to 90 in 30 minutes.

With the cooperation and assistance of Dr. Harvey J. Carlson, Assistant Professor of Pediatric Research, the following study was made to determine by *in vitro* techniques whether the Duponol-C in the concentrations suggested has any specific action on respiratory mucin.

PURPOSE.

The purpose of this study was to investigate the action of 0.1 per cent Duponol-C solutions in reducing the surface tension, viscosity, and fluidity of respiratory mucin, and to determine the effect of Duponol-C on animals when administered by the intranasal route and by nebulization. The effectiveness of Duponol-C in increasing humidity was also studied.

METHODS AND EXPERIMENTAL RESULTS.

HUMIDITY.

A number of government weather bureau stations now report humidity in grains per pound instead of relative humidity. This is the actual amount of moisture in a given amount of air. This is actual or absolute humidity. It is a figure which is independent of the temperature of the air. It thus has more meaning for comparing figures. This value is obtained by calculation from the dew point, which is the temperature at which the air would become saturated if it were cooled. The dew point is determined by readings of a wet and dry bulb thermometer.

It is true that scientific and experimental reporting of amounts of humidity would be more accurate for purposes of comparison if such data were reported in grains of moisture

per pound. Relative humidity figures are significant for comparison only when the dry bulb temperatures are the same.

For this study a room was used which had been prepared especially to maintain high humidity levels. The room contained approximately 4,000 cubic feet. It had one sealed window. The walls were painted with a special moisture resistant paint. A vapor barrier at the door consisted of a felt strip sealing the edges. There were no drapes or carpets in the room.

A Walton humidifier containing tap water only was placed in the room and started in operation. Wet and dry bulb readings were made every 15 minutes until three successive readings remained constant. This point was reached in one and one-half hours. Duponol-C was immediately added to the water in the humidifier in the concentration of 0.1 per cent and the readings were continued every 15 minutes for another one and one-half hours. The wet and dry bulb readings were the same following the addition of the Duponol-C and remained constant for the full one and one-half hours. The dew points, once a constant level was reached, remained unchanged.

A similar test was made in the same room using two large steam vaporizers. With water only in the vaporizers, wet and dry bulb readings were made every 15 minutes until three consecutive readings were the same. This point was reached in two hours. Again Duponol-C was added to each vaporizer in the concentration of 0.1 per cent and the readings continued at 15-minute intervals for two hours. There was no change in the wet and dry bulb temperatures or dew points following the addition of the Duponol-C.

Duponol-C is the trade name for the chemical sodium lauryl sulfate, a salt of a higher alcohol. The Du Pont laboratories[15] report that only some of the fatty acids would come over in the steam vapor. The chemical would be present, however, in the particles of water dispersed from a Walton cold type humidifier.

SURFACE TENSION.

The duNouy tensiometer employing the ring method and the capillary height method were used to determine surface tension. The ring method was fast and the accuracy sufficient for the study. The capillary height method was used only to check the ring method tensiometer.

The capillary bore size was determined by using mercury and water. We used the equation $S = \frac{h\,g\,d\,r}{2\,COS\,\Theta}$ (S is surface tension in dynes/cm., h is capillary rise of fluid in cm., g is acceleration due to gravity, d is density, r is the radius of the tube), which could be simplified to one-half h d g r because Θ, the angle of contact, was so small it was always practically one. The diameter of the capillary was found to be 0.06 mm. using water or mercury. Surface tension of a 10 per cent gastric mucin solution was determined to be 49.0 dynes per cm. by the capillary height method and 48.0 dynes per cm. by the duNouy tensiometer. These findings were in very close agreement, so that the remaining determinations were made with the duNouy ring tensiometer to facilitate greater numbers of determinations.

Two ml. of each of the solutions listed in Table 1 were placed in a watch glass and six to 10 surface tension determinations were made. The figures listed are the average of six or more readings in one series of tests made. The original respiratory mucin was tenacious and was mixed in a Waring blender with saline 1:1. The resulting solution was homogenous and was used as the concentrate.

The data presented in Table 1 show that the Duponol-C solutions lower the surface tension of both the 5 per cent gastric mucin and the concentrate respiratory mucin when mixed in equal proportions. There was an 11 and 11.5 dyne difference (23 per cent) in both mucins when 1 per cent Duponol-C was used as compared to water. The difference was slightly less when 0.1 per cent Duponol-C solutions were used.

TABLE 1. SURFACE TENSIONS OF MUCIN SOLUTIONS.

No.	Solution	Surface Tensions* 15 min. (A)	6 hr. (B)	24 hr. (B)	Remarks
1	Water	71.5			Distilled
2	Duponol	33.5			1.0 per cent
3	Duponol	33.5			0.5 per cent
4	Duponol	33.5			0.1 per cent
5	Duponol	32.5			0.05 per cent
6	Gastric Mucin	48.0			10.0 per cent
7	Gastric Mucin	42.0	45.0	48.0	5.0 per cent
8	Gastric Mucin	45.0	45.0	47.0	2.5 per cent
9	Resp. Mucin	54.5	49.5	46.5	Concentrate
10	Resp. Mucin	54.5	53.0	50.0	Concentrate and distilled water 1:1
11	Gastric-Duponol	33.5	32.5	32.0	5% gastric mucin and 1% Duponol 1:1
12	Gastric-Duponol	33.5	32.5	32.0	5% gastric mucin and 0.1% Duponol 1:1
13	Resp.-Duponol	33.5	33.0	32.0	Resp. mucin (Conc) and 1% Duponol 1:1
14	Resp.-Duponol	39.5	39.5	39.5	Resp. mucin (Conc) and 0.1% Duponol 1:1

* Dynes per cm.
(A) 25° C.
(B) 37° C.

VISCOSITY.

The viscosity of the solutions was measured with the modified Ostwals viscosimeter. As the measurements were preliminary, all readings were made at room temperature. One viscosimeter, calibrated for 32 seconds with double distilled water, was used for all determinations. The average flow time of distilled water through this tensiometer at room temperature (27° C.) was 33.5 seconds, computed after numerous determinations. Table 2 presents the viscosity of solutions of gastric and respiratory mucin. The gastric mucin was prepared in a basic 10 per cent concentration. Dilutions were made from this solution and mixed with varying concentrations of Duponol-C. The respiratory mucin had previously been diluted with 1:1 saline and thoroughly mixed in a Waring blender to obtain a homogenous solution. The viscosities

TABLE 2. VISCOSITY AND FLUIDITY OF MUCIN SOLUTIONS.

No.	Solution	Dilution	Sec. (Aver.)	Viscosity*	Fluidity†	Remarks
1	Water	—	33.5	1.00	100.0	Distilled
2	Duponol-C	10^{-2}	38.6	1.14	87.7	Prepared in distilled
3	Duponol-C	10^{-3}	35.9	1.07	93.4	Prepared in distilled
4	Gastric Mucin	10^{-2}	56.9	1.69	59.1	
5	Gastric Musin	2×10^{-2}	43.5	1.29	77.5	0.5% gastric mucin
6	Gastric Mucin	10^{-3}	38.2	1.14	87.7	
7	Respiratory Mucin	—	139.0	4.52	22.1	Concentrate—Original solution and water (1:1)
8	Respiratory Mucin	1:1	57.0	1.70	58.8	No. 7 and water (1:1)
9	Gastric Mucin—Duponol	1:1	52.4	1.56	64.1	1.0% gastric mucin and 1.0% Duponol (1:1)
10	Gastric Mucin—Duponol	1:1	50.7	1.51	66.2	1.0% gastric mucin and 1.0% Duponol (1:1)
11	Respiratory Musin—Duponol	1:1	71.1	2.12	47.1	Concentrate respiratory and 1.0% Duponol (1:1)
12	Respiratory Mucin—Duponol	1:1	58.2	1.73	57.8	Concentrate respiratory and 1.0% Duponol (1:1)

*Coefficient of viscosity—(c. g. s. units—poise)—centipoises.
†Fluidity = rhes.

of the solutions were determined from the following equation: $\frac{n_1}{n_2} = \frac{d_1 t_1}{d_2 t_2}$ where n_1 and n_2, d_1 and d_2, t_1 and t_2 are the viscosities, densities and times of efflux, respectively, for the two liquids. The fluidities of the various liquids are also shown in Table 2 and are expressed in rhes.

As shown in Table 2, the viscosity of water and the Duponol-C solutions were approximately the same. Gastric mucin was used as a control solution since known concentrations could be prepared. The viscosity of a 1 per cent solution of gastric mucin was not significantly altered when mixed with water 1:1, making a 0.5 per cent solution. The addition of either 1 or 0.1 per cent Duponol-C solution reduced the viscosity 0.2 poise. The difference was so small that it cannot be considered significant. Respiratory mucin was found to have a viscosity of 4.52 and when diluted 1:1 was 1.70. Mixtures of respiratory mucin was 0.1 per cent Duponol-C were found to have similar viscosities to those of water-respiratory mucin solutions. One interesting finding was the higher viscosity obtained using 1 per cent Duponol-C solutions. From the other results, the viscosity should be similar to that obtained with the less concentrated solution.

PENETRATION.

Table 3 shows the results of preliminary tests to determine the possible mucolytic action of Duponol-C. This solution

TABLE 3. MUCOLYTIC ACTION.

No.	Solution	1.0% Duponol		Water		Remarks
		Penicillin	Untreated	Penicillin	Untreated	
1	10% Gastric	—	—	—	±	Prepared with water
2	10% Gastric	—	++	—	+++	Prepared with phosphate buffer
3	5% Gastric	—	++++	+	++++	Prepared with phosphate buffer
4	Respiratory	+++	++	—	—*	*Partial gray—bacterial?

(1 ml.) was layered over (2 ml.) mucin. Gastric mucin and water were used as controls. One group of tubes was mixed with penicillin to control bacterial action. The tubes were incubated at 37° C. for 18 hours.

The gastric mucin prepared with phosphate buffer was black in color and liquefied in the tubes which were not treated with penicillin. Both Duponol-C and water gave simlar results. The gastric mucin was sufficiently acid to control the bacterial breakdown. There was no effect of the water on the respiratory mucin in either tube.

Both of the tubes containing respiratory mucin, one with penicillin and one not treated, were liquefied. There were some small pieces of undissolved mucin, but the majority was liquid in character.

TOXICITY.

Duponol-C solution (0.1 per cent) was given intranasally to partially anesthetized mice and also nebulized in a closed chamber for two hours to a second group of mice. These animals were sacrificed at regular intervals, and the lungs were removed and prepared for sections. Unfortunately the specimens have not been received from the pathological laboratory, and the findings cannot be given. These will be added at a later date.

COMMENT.

It is apparent that the addition of 0.1 per cent Duponol-C to vaporizers or humidifiers in the treatment of acute laryngotracheobronchitis will not increase the relative humidity or the absolute humidity. Any beneficial effect must be the result of its action on the respiratory secretions.

The action of Duponol-C on the two types of mucin used in the present study was negligible except for the findings on surface tension and penetration (mucolytic). The findings on surface tension could be due to dilution alone except for the results with water. The surface tension of water was high and did raise the surface tension of mucin mixtures mater-

ially. The Duponol-C solutions reduced the surface tension to their own levels. If it was dilution only, it seems there should have been some increasing of the surface tension measurements with Duponol-C. Other factors undoubtedly play a very definite rôle in this action. To properly evaluate this specific phase of the problem, an interfacial tensiometer should be used.

The test made for penetration or mucolytic activity was made on a different sample of respiratory mucin from the rest of the tests. This mucin was so tenacious and thick that it could be removed only by cutting with scissors.

The gastric mucin was definitely liquefied by the action of bacteria, as only those of neutral pH or not containing antibiotic became dark in color and liquefied. The respiratory mucin was not affected by the water or the possible presence of bacteria. The respiratory mucin in the presence of 1 per cent Duponol-C was greatly changed and was liquid.

The tests described in the preliminary report indicated that some effect was obtained. Repeated determinations will have to be made using freshly collected respiratory mucin. Changes in viscosity and fluidity should demonstrate measurable differences.

I regret the inability to report at this time the pathological reports of the lungs of the mice exposed to Duponol-C by nebulization and intranasal instillation. Hall[10] reported the pulmonary toxicity of several wetting agents, including Duponol-C in concentrations of 0.1, 0.5 and 1 per cent aerosols on guinea pigs exposed eight hours a day for six days. All animals exposed to the 1 per cent concentration were very dyspneic, many died, and all showed severe lung reactions. Those exposed to 0.5 per cent concentrations showed mild to moderate dyspnea and mild lung reactions. None of the animals exposed to the 0.1 per cent concentration showed any dyspnea or lung reaction. The reactions when exhibited consisted of polymorphonuclear cells and macrophages, proliferation of fibroblasts of septal walls, degeneration and desquamation of

pulmonary epithelium and proliferation of alveolar epithelium. In large areas the alveoli were completely obliterated by this reaction.

SUMMARY.

The action of Duponol-C and water on gastric and respiratory mucin was observed.

Surface tension, viscosity, fluidity and penetration (mucolytic action) measurements were made.

The effect on humidity of the addition of Duponol-C to water in a humidifier or a vaporizer was measured and found to be negligible.

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DR. HALLOWELL DAVIS WINS OTOLOGY PRIZE.

Dr. Hallowell Davis, director of research at Central Institute for the Deaf, St. Louis, has been awarded the George E. Shambaugh prize in otology—by the Collegium Oto-Rhino-Laryngologicum Amicitae Sacrum. He is the first American scientist to receive the honor.

Dr. Davis was presented with $1000 award "as a recognition for outstanding work on the physiology of hearing and the development of testing procedures for hard of hearing individuals." The Collegium is an international society devoted to scientific study of the ear, nose and throat.

The Shambaugh prize, established in 1947, is awarded every three years to the scientist selected as having made an outstanding contribution to knowledge of hearing and deafness.

CLASSIFICATION OF CARCINOMA OF THE LARYNX.

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Much literature on cancer of the larynx has been collected in a relatively recent period. This disease was but vaguely known in olden times. In 1790 Morgagni[19] referred to two of Valsalva's cases, in his Anatomical Letter No. 28, in his book "De sedibus et causis morborum per anatomen indagatis"; these were actually laryngo-pharyngeal carcinomas.

In 1833, Urner[23] published two cases and Albers[1] one, of carcinoma of the larynx. In 1834, Trousseau performed a tracheotomy in a case which he studied very carefully in postmortem examination, the specimen having been examined by Cruvelier. The complete report was published by Trousseau and Belloc[22] in 1837. During the same year, Louis[16] published a detailed observation which he thought to be the first registered in medical literature.

With Garcia's invention of the laryngoscopic mirror, observations became more frequent. E. Blanc[2] published a thesis in 1872 and Fauvel[6] made a detailed report of 37 cases in 1876.

Cancer of the larynx is no longer a rare disease. The successive contributions in the clinical, surgical, anatomical, pathological and radiological fields have increased our knowledge and experience; although the former is still limited. One of the urgent needs of research undertaken to improve methods of treating cancer, is the adoption of uniform classification and staging of the disease in the common sites. Unless facts and observations can be classified by a method capable of similar application by different observers, it will be impossible to understand the behavior of the disease during various methods of treatment.

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Should an international agreement be reached as to nomenclature and classification of cancer of the larynx, both in relation to the topographical site and to the different degrees of anatomical-clinical extent, a formidable series of comparable clinical material could be obtained, allowing appreciation of the efficiency of the various methods.

It is difficult to establish a correct classification of carcinoma of the larynx. The idea is by no means new, it dates from the dawn of the laryngoscopic era, when a small number of cases of cancer of the larynx were observed. From then to the present day, little improvement has taken place and the time has come to try to achieve a convenient classification, although perfection is not likely to be reached. The first difficulty arises when an attempt is made to find a topographical definition of cancer of the larynx, because the anatomists ascribe to the larynx, regions which do not belong to it exclusively.

The laryngeal region, is a hollow pyramid with an inferior vertex and two antero-lateral walls with precise boundaries. The base of the pyramid and the posterior wall also form part of the pharynx.

In 1866, Krishaber[10] distinguished carcinoma arising in the base and posterior wall from those arising within the laryngeal cavities and designated as laryngeal cancer only the endolaryngeal lesions.

In 1876, Fauvel follows a similar system and classifies them as:

1. Primary cancer of the larynx;

2. Mixed cancer; meaning those that straddle on the larynx and pharynx;

3. Secondary or neighboring cancer, or consecutive esophago-laryngeal cancer.

Isambert[9] did not agree with Krishaber's idea and did not see why tumors developing in the opening and in the pharyngeal wall of the larynx, should be excluded, naming them "extrinsic" or laryngeal carcinomas.

It is here that the term "extrinsic cancer" appears for the first time, applied by Isambert with the remark: "We use here the word extrinsic in the same sense of the anatomical expression, extrinsic muscles of the larynx. They are not carcinomas strange to the larynx but carcinomas inserted in the exterior walls of the organ which may extend to the neighboring organs."

Isambert completed this classification in the following way:

1. Extrinsic cancer, or laryngo-pharyngeal;

2. Polypiform intra-laryngeal cancer;

3. Scirrhous intra-laryngeal cancer.

Oddly enough the classification of laryngeal cancer in extrinsic and intrinsic, is attributed to Isambert in the French medical literature and to Krishaber in the English and American.

In 1879, Krishaber[11] published a second work on cancer of the larynx considering Isambert's criticisms, he agreed that in view of the similarity of their symptoms and treatment to those of the endolaryngeal cancer, some laryngo-pharyngeal cancers may be included with the primary cancers of the larynx.

Nevertheless, the majority of contemporary authors accept Krishaber's first classification and that is also our own choice.

Further on, this author continues: "In truth, we are before two varieties, the intrinsic and extrinsic cancer. The first is implanted and is developed in the cavity proper to the organ, the second on the walls; the first variety includes Isambert's sub-glottic or tracheal cancer, a rare form of which I have observed one example. Carcinomas of the second variety, those called extrinsic or mixed, that develop in the epiglottis, at the superior orifice of the larynx, on the arytenoid wall, are more frequent than intra-laryngeal cancers, but it is important to know by what characteristics they enter into our study and by what differences they are separated from those carcinomas occupying the cavity". It is evident that

the word *extrinsic* was used for the first time by Isambert, and the word *intrinsic* was used for the first time by Krishaber, a divided responsibility which has led some authors to give either to Isambert or to Krishaber the credit for classification and for the terminology of extrinsic and intrinsic.

This classification, more than 70 years old, continues to be applied by many authors, but in different ways, thus leading to confusion. Trotter,[21] in 1913, called intrinsic cancer only those of the vocal cord and subglottic areas. Hayes Martin[17] follows very closely this criterion and calls extrinsic those originating in the free border of the ventricular band, including marginal and retrocricoid cancers and excluding those of the lateral and posterior walls of the pharynx and pyriform sinus.

As a result of modern experience, many authors have tried to distinguish primary sites in the different structures of the endolarynx and have classified them as belonging to the laryngeal wall of the epiglottis, ventricular band, ventricle, vocal cord, subglottis, etc. Others have referred to the anatomical division into regions of the endolarynx: supraglottis, glottis, subglottis; the classification followed in general by Hautant, Leroux-Robert, Baclesse, Lederman and myself. My first object is to suggest a classification according to the initial site based on Krishaber's first classification and including certain convenient terms used by other authors. I believe that confusion will be avoided if extrinsic and intrinsic are discarded.

In addition to classification I shall suggest a method for staging cancer of the larynx according to the anatomical-clinical extent.

The primary idea of such a classification is that it would form the basis for a more detailed grouping by localization e.g.: epiglottis, ventricular band, ventricle, anterior or posterior segment of the vocal cord, anterior or posterior commissure etc., as to the different clinical macroscopic groups: exofitic or papillar, infiltrative, excavating or to histo-pathological nature.

All these characteristics are interesting, but it would be too complex to include them in a basic classification, for they are often difficult to determine accurately and also many sub-divisions lead to so few cases in each, that statistical analysis loses its value; nevertheless in the future as numbers increase, sub-divisions can be added.

The following classification is, therefore, the simplest possible; it groups according to primary site and gives certain criteria for staging.

REQUIREMENTS OF A SOUND CLASSIFICATION.

1. That it should be simple; easy to understand and to apply.
2. Classification should be as precise as possible.
3. It should define four stages definitely described by anatomical and pathological findings obtained by ordinary clinical examination.
4. As site already gives three groups, stage should be limited to four stages, so that each stage may contain a number of cases sufficient for a statistical evaluation of the results.

NOMENCLATURE.

Carcinoma of the Larynx. Number of the International Classification of the disease: "161".

Definition of the Larynx.—Larynx includes the entire endolarynx. The upper border of the larynx is formed by the epiglottis, the ary-epiglottic folds and the ary-tenoid region, (inlet or crown of the larynx). The inferior limit of the larynx is at the level of the lower border of the cricoid cartilage. Carcinomas arising in this border-line could belong to the pharynx.

Different sites of Carcinoma of the Larynx.—Carcinomas of the larynx are classified into three groups:

1. Carcinomas of the supraglottis.
2. Carcinomas of the glottis (vocal cord).
3. Carcinomas of the subglottis.

Staging of clinical material according to the extent of the growth for Carcinomas of the Supraglottis, Glottis and Subglottis.

Stage I—The carcinoma is limited to the mucous membrane. No infiltration. Full mobility of the larynx is retained.

Stage II—The carcinoma infiltrates but does not extend beyond the larynx. Mobility of the larynx is impaired or lost.

Stage III—The carcinoma has extended beyond the larynx, direct extension or lymph node metastasis but without producing the conditions specified in stage IV.

Stage IV—Involvement of skin or fixation of metastatic node or distant metastasis.

Note: If any doubt exists the cases should be classified in the immediate lower stage. The presence of more than one characteristic does not modify the stage.

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THE PROBLEM OF THE BLUE EARDRUM IDIOPATHIC HEMOTYMPANUM.*

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INTRODUCTION.

The term, idiopathic hemotympanum, has been used occasionally to designate a condition which has attracted all too little attention in the medical literature. Referred to by some as "the syndrome of the blue eardrum", the term alludes to an unexplained startling color of the membrana tympani, described by various authors as "steel-blue", "blue-black", or "indigo". If the paucity of pertinent literature can be considered indicative, the condition is unusual if not rare, although there may be reason to suspect that it would be more frequently reported if more adequately publicized. For example, another possibly related disease, serous otitis media, has been recognized since about 1860, but only in recent years has it received the attention it deserves. There are less than a dozen direct references to hemotympanum in the medical literature, and most of these mention the phenomenon only as a puzzling curiosity.

For generations, the elusive color of the human eardrum has defied all efforts to describe it adequately and has inspired one elaborate monograph which has become a historical classic. Politzer[14] in 1869 demonstrated his remarkable powers of observation in his treatise, "The Membrana Tympani in Health and Disease". He mentioned a "bluish tinge" to the pearly appearance of the normal drum when viewed by daylight and commented at length upon the effect of light-source upon the apparent color of the drum as seen by the observer. He even discussed serous otitis media with an understanding which must have stemmed from his own

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independent observations and thinking; but nowhere did he mention the blue eardrum as an entity. The earliest reported case would seem to be Gruber's[19] in 1888, and then probably Kerrison's[10] mention of hemotympanum in 1914. The first serious consideration of idiopathic hemotympanum as an actual disease entity was by Shambaugh[17] in 1929. Prior to this time, all collections of blood in the middle ear were considered to be the result of obvious trauma or anomalies of the tympanic floor with protrusion of the jugular bulb. Shambaugh's case reports apparently stimulated interest in the subject, and although subsequent writers have not necessarily accepted his theories, they consistently refer to his basic observations. Ranger[15] in 1949, and finally Vior[19] in 1951 have attempted to stimulate interest further by their case reports and interesting speculation regarding possible causes of the disease.

In recent years, both medical and public attention have been increasingly directed to problems of hearing loss, particularly in children. Inevitably, the syndrome of the blue eardrum becomes a part of this problem, albeit a minor part from the numerical standpoint. Authorities consistently have noted the apparently disproportionate amount of conductive deafness present in these cases, and the persistence of the loss in untreated cases. In a few recorded instances, there has been a disappointing lack of improvement even in those treated to the best of our present knowledge. It is more than likely that many cases of hemotympanum are not seen by otologists and perhaps not even by any physician. The insidious and painless character of the disease lends itself to neglect by unobservant patients to whom a mere dull sensation in one ear might not indicate need for medical attention.

Because of our limited experience with this unusual cause of deafness we are by no means satisfied that all the answers to its cause and its treatment are at hand. No single observer has had the opportunity of studying and treating enough cases to formulate these answers. Only by pooling our aggregate experience, however scant, will we bring

adequate information to bear upon solution of the problem. Thus far, we must admit to little more than speculation and empirical trial and error.

KNOWN CAUSES OF BLUE EARDRUM.

The presence of blood in the middle ear has probably been observed many times by every otologist in consultation on cases of head-injury, particularly of basal skull fracture. It is well known that even without visible evidence of injury to the membrana tympani or the annulus, the tympanum may be found to be completely filled by blood under sufficient pressure to cause bulging of the drum or leakage into the pharynx through the Eustachian tube. We have, as yet, never seen the hemorrhagic fluid fail to absorb and disappear within a month or six weeks. Even in instances of fracture with cochlear or acoustic nerve damage, resulting in total perceptive deafness, the drum assumes its normal color and appearance after a reasonable interval. Dickson[3] reported his experience in testing cadets for susceptibility to barotrauma. The decompression chamber was used to simulate rapid descent in military aircraft, with resultant rapid increase in barometric pressure. The great majority of the cadets were able to equalize their middle ear pressure adequately either by conscious effort or by subjective automatic responses. Of eighty-nine cadets who showed objective signs of barotrauma, as indicated by retraction or congestion of the eardrum, fifteen developed an effusion into the middle ear, and one developed an actual hemotympanum. He states that, without exception, the middle ears containing fluid had completely cleared themselves within fifteen hours. The same author[2] in experimental work with cats, was able to produce hemotympanum only by "explosive" reduction of barometric pressure in the chamber. These experiments were conducted upon thoroughly narcotized animals in which any protective equalizing reflexes were obviated.

Vior[19] enumerated, for the sake of completeness, several unusual causes of blue eardrum with definite physical lesions demonstrable at operation or at autopsy. He referred to

cases of hemangioma in the middle ears of children as reported by Halphen. Similarly, Tempea[19] cited a case of actual varices in the middle ear. He further referred to Richmann's experience with a similar instance of angiomata in the tympanum which revealed dark blue "spots" in separate portions of the drum. Sheppard[18] described a hemophilic child with recurrent bilateral hemotympanum, and Burgois and Escat were cited by Vior again as reporting hemotympanum in several hemophiliacs. By analogy, hemorrhage into the middle ear could be expected as a complication of any hemorrhagic diathesis.

The early writers who mentioned a blue eardrum seem to have been obsessed with the idea of the jugular vein as a causative agent. This is, no doubt, a reasonable idea when we compare the color of the blue eardrum with the color of the juglar vein or the lateral sinus as observed during surgery. The striking blue-black color with an overlying dull gray sheen is very similar in the two instances. Early disastrous experience as described by Gruber,(Vior, 19) Phillips,[13] and Harris,[7] with myringotomy into the jugular bulb, or with mastoid surgery in the face of almost uncontrollable hemorrhage, led some observers to assume that blue eardrum, otherwise unexplained, was always the result of a jugular bulb protrusion into the tympanum. Phillips[13] described a huge accessory petrosal vein located just posterior to the promontory, observed during a radical mastoid operation. Doubtless such anomalies do occur, and presumably could give a blue color to the eardrum. Osteologists describe variable but appreciable percentages of specimens showing dehiscences in the floor of the tympanum. Korner, Budder, and Muller(Vior, 19) found two to six per cent of such defects in skulls which they studied, and others have reported even higher figures. The defects vary from minute channels which could contain collateral venules, to large apertures through which a jugular bulb could easily protrude. Vior feels that Gompez, Frimach, and Salgmann are probably correct in maintaining that herniation of a jugular bulb through even a large defect in the tympanic floor must presuppose the existence of a concurrent weakness in the wall of the bulb.

Such a weakness could be the result of increased venous pressure from any cause, or the result of inflammation or toxic disease of the wall of the bulb itself. Kerrison[16] mentioned Dench's[1] experience in exposing the jugular bulb while performing an ossiculectomy. Kerrison himself described cases in which surgery or necropsy revealed destruction of the tympanic floor by infection or cholesteatoma.

THE IDIOPATHIC BLUE EARDRUM.

The earliest description of the idiopathic blue eardrum was in 1914 by Sheppard[18] who mentioned this peculiar phenomenon during his discussion of hemophilia with concurrent hemotympanum. He seemed to realize that it was something unusual and commented upon the absence of discernible cause, in one of his cases, in contrast to those cases in which hemophilia was demonstrated to be present. He apparently assumed, in the light of current knowledge, that he was dealing with a jugular bulb protrusion, and abstained from myringotomy.

Shambaugh[17] in 1929 described the blue eardrum in which no local or systemic cause was evident. Apparently he was the first to recognize this type of lesion as an entity. He described the typical color involving only the pars tensa and quite frankly admitted that he was not satisfied with any explanation. His careful investigation included repeated myringotomy and an excellent description of the curious syrupy brownish fluid which consistently gave negative cultures for any known organisms. He likewise performed repeated inflations with disappointing results. He recalled having seen such a case in 1920 but had regarded it as a curiosity of little significance until his contact with similar cases nearly ten years later. He advanced three possible causes which he admitted leave much to be desired by way of explanation: *1.* rupture of a hemorrhagic bleb in acute otitis media; *2.* protrusion of the jugular bulb through a dehiscence; and *3.* Eustachian tube obstruction. Exploration of the mastoid was not mentioned although he noted roentgenological changes on the involved side.

In 1939, Fowler[6] mentioned a case of blue eardrum, and described the appearance fairly well. He commented upon the surprising degree of conductive hearing loss with minimal symptoms of discomfort, but drew no conclusions.

Kler[11] in 1948 reported an apparently typical case in an adult. He was able to effect a partial cure by repeated myringotomies and inflations, but in spite of improved hearing, part of the blue color returned to the drum. He concluded that tubal obstruction was the underlying cause but did not mention mastoid X-rays.

The first approach to the problem with a serious effort to exhaust the diagnostic possibilities was by Ranger[15] in 1949. He presented two cases with physical findings strikingly similar to those described by the earlier writers. With the experience of these men in mind, he systematically tried inflations, repeated myringotomies, and finally, encouraged by X-ray findings of mastoid pathology, he felt justified in exploring the mastoid. In both cases he found the cells filled with the identical brownish, syrupy, sterile fluid he had obtained by myringotomy. Laboratory examination of the fluid indicated that it was essentially altered blood. Pathological examination of bone from the mastoids indicated no destructive infectional process but rather cholesterol clefts from blood deposition. Neither of his patients showed laboratory evidence of any blood dyscrasia or capillary abnormality to explain hemorrhage into the middle ear and mastoid. It is noteworthy that his first patient responded by satisfactory improvement in hearing, but that his second was complicated by an apparently unrelated nerve deafness and failed to improve following surgery. His discussion and comments are impartial and thought provoking. He systematically eliminated the simple explanations for the condition by citing exceptions to the rules, and concluded that no single condition could be implicated as the entire cause. He admitted that O'Donnell[12] had demonstrated Eustachian tube obstruction as the cause in his two cases, but in Ranger's own experience tubal obstruction seemed to have no bearing. He visualized the cellular mastoid as a reservoir for the sero-

sanguineous material, constantly replenished by repeated fresh hemorrhages whenever he removed fluid by myringotomy and thereby reduced pressure in the middle ear. Although he found no source of unusual bleeding in the operated mastoids, he decided that exenteration of the cells must have obliterated some such occult source which was not grossly visible.

The most recent report of the blue eardrum comes from Madrid, Spain. Vior[19] describes vividly his observations on a case in 1951. In spite of unavoidable loss of descriptive precision in translation from the original Spanish, his observations are astute and show a keen appreciation of the fact that he was confronted by an uncommon disease. He found no evidence of tubal obstruction, but observed the blue color in his case to be confined to the postero-inferior aspect of the drum, and to pulsate as if in contact with a major vessel. He reviews the literature and concludes that his case must be classified as one of jugular bulb protrusion. He recognizes the existence of an idiopathic form of hemotympanum, but believes that before even diagnosic aspiration or myringotomy is attempted, the likelihood of jugular encroachment must be ruled out so far as possible. He outlines an excellent course of differential diagnosis, and decided in his own case to depend upon treatment of the patient's "nasal catarrh" rather than risk incision of the jugular bulb.

DIFFERENTIAL DIAGNOSIS.

As can be readily inferred from the dire warnings of our early otologists, and from the more recent observations of Vior who conservatively refrained from surgical interference in a case of blue eardrum, that differential diagnosis presents a problem of critical importance. Incision of a major venous anomaly or a jugular bulb would be most undesirable if not disastrous. On the other hand, the loss of hearing incident to hemotympanum would seem to be both severe and permanent, unless adequate drainage is established and the middle ear evacuated.

Trauma and constitutional hemorrhagic diatheses are readily eliminated as causative factors by virtue of concurrent

symptoms and signs, history, and laboratory findings. In the former instance, absorption removes all or most of the hemorrhagic result of injury to the middle ear. In the latter instance, absorption may take care of isolated episodes of intra-tympanic hemorrhage, but the future of a hemophiliac will continue to be hazardous regardless of any known form of treatment. It is entirely reasonable to expect hemotympanum occasionally to complicate the various forms of purpura, but we have seen no reports of such findings.

Tumors of the middle ear producing a blue eardrum from hemorrhage would be difficult to eliminate unless there was X-ray evidence of bone-destruction, or extension into the canal. Reports of angiomata and varices in the middle ear leave much to be desired in regard to accurate description. Richmann[Vior, 19] described the discrete blue areas visible through the membrana tympani in his case of angioma, but it is conceivable that a uniform coloration and probably even bulging might equally well appear. In any event, cases in this category would probably be surgically explored on the basis of X-ray findings.

Protrusion of the jugular bulb into the tympanic cavity seems to produce variable degrees of change in the color of the eardrum. Several early reports of such findings at surgery or autopsy fail to mention any peculiar color of the drum. Vior's case showed the typical color only in the inferior portion of the drum and, probably the most important diagnostic sign, showed definite pulsation. There was also a pronounced bulge which increased in this blue area when the homolateral jugular vein was manually compressed. Inflation produced no objective or subjective change in Vior's case, and produced neither a fluid level nor bubbles in the middle ear. It is noteworthy that the authors who inflated ears with idiopathic hemotympanum observed shifting areas of blue color or a definite fluid level, and also observed improvement in the subjective sensation of obstruction and deafness. Apparently inflation and change of position of the head can temporarily free the foramen rotundum from

obstructing fluid, as has been similarly noted with serous otitis media (Hoople,[8]). All reported cases of idiopathic hemotympanum in which X-ray studies have been done show unmistakable evidence of mastoid pathology, consisting of at least blurring of cell-margins and sometimes actual bone destruction.

Amalgamation of the foregoing observations and opinions from diverse sources would indicate a method of differential diagnosis approximately as follows:

1. Meticulous examination of general ear, nose and throat system, including nasopharyngoscopic examination and sinus evaluation.

2. Careful observation of the eardrum for color, distribution of color, bulging or retraction, fluid level or bubbles, and pulsation.

3. Careful audiometry with determinations of air and bone conduction.

4. Inflation of middle ear and recheck appearance of membrana tympani. Check for shifting fluid level with change of head position and for bubbles.

5. Observation of the involved eardrum with the Siegle otoscope and with manual pressure upon the homolateral jugular vein.

6. Mastoid X-rays to determine changes in the cellular structure or possible bone invasion or absorption in the tympanum.

7. Rule out constitutional causes by complete physical examination and pertinent laboratory procedures. Obviously this should include bleeding and coagulation time, platelet count, and capillary fragility tests. When the observer is sufficiently satisfied that he is dealing with free fluid in the middle ear and not a hazardous anomaly, he should obtain a specimen of the fluid by aspiration or myringotomy. In many instances, a second aspiration puncture in another quadrant of the drum (Hoople,[8]) will assist in withdrawal

of particularly viscid fluid. An actual myringotomy followed by inflation and point suction with a small tip is more likely to be successful. The number and variety of laboratory procedures justifiable with regard to the fluid is a debatable issue. Ideally, aseptic technique should assure a reliable culture report. Microscopic examination for formed elements has not proven particularly helpful in reported cases. A van den Bergh test and quantitative examination for cholesterol will serve only to suggest that the fluid is hematogenous in origin. Its gross chocolate appearance is practically diagnostic of this peculiar condition and, in the light of experience thus far, obviates laboratory tests for all practical purposes. We have all observed the viscid reddish-brown fluid aspirated in considerable quantity from old subcutaneous hematomata. Sometimes, we should recall, this material is quite liquid, and at other times its viscosity may necessitate use of perhaps a sixteen gauge needle for aspiration. The appearance of fresh blood upon myringotomy is not to be expected in these cases and must be considered indicative of a venous anomaly or an angiomatous tumor. For purely investigative purposes, any laboratory tests conceivably pertinent are in order, but, because of their doubtful diagnostic value, the otologist will probably be guided by the economic factors involved.

TREATMENT.

Although use of the term "idiopathic" is abhorrent to many medical writers, its non-committal connotation has a certain value in regard to the syndrome of the blue eardrum. The term "idiopathic hemotympanum" is at least descriptive and, in view of the foregoing experience and opinion from diverse sources, is probably justified. Until more is known of the true origin of this interesting condition, a more accurate nomer is out of the question. It would seem that the only common ground for opinion on the subject is in the field of clinical description; and, meager though the ventures into conjecture on possible causes may be, only one author has actually followed up his reasoning with definitive action. Ranger[15] concluded that the mastoid cavity must be acting

as a reservoir for this fluid of hematogenous origin. He reasoned that even though the actual point-source of the recurrent hemorrhages might be in the Eustachian tube or the tympanum, removal of the relatively enormous reservoir capacity of the mastoid cellular structure must be necessary if a cure is to be expected. Thus, of the cases of idiopathic hemotympanum reported in the literature, only two have been subjected to surgical exploration. With this limited precedence for support, no one can safely outline any routine course of treatment. Apparently no urgent necessity for surgical intervention can be considered to exist. All the reported cases have been known to present the diagnostic findings for varying periods of time up to five years or even more. The condition is apparently not self-limiting, nor is it likely to terminate spontaneously, but thus far it has never become a surgical emergency; therefore, the universal attitude of conservatism is fully justified up to the point at which no progress is evident. Ranger was not satisfied to turn aside at this point and consequently suggested the course to follow when conservative measures fail.

Once a diagnosis is certain and the presence of the typical fluid in the middle ear is established, treatment should reasonably be expected to start with the most conservative procedures. O'Donnell's[12] success with inflation would imply that there are cases which will respond thus. Obvious tubal obstruction by gross lymphoid tissue is self-evident and must be eliminated perforce. Even the method of inflation is the subject of some difference of opinion; probably either catheterization or the Politzer method will prove equally efficacious. If several adequate inflations with some objective signs of visible bulging of the drum or bubbles in the middle ear are not effective, repeated myringotomy or aspiration is the next step to initiate. Most of the authors cited have resorted to myringotomy, and report variable periods of time for the fluid to re-accumulate, as it almost certainly will. After several thorough evacuations of the middle ear by this method, there must be evidence of decreasing re-accumulation as demonstrated by longer intervals between necessary myringotomies and by decreasing amounts of

fluid obtained, or evacuation must be considered only a temporary expedient. If Ranger's limited experience can be considered a guide, mastoidectomy must be contemplated in the event of such failure. X-ray or radium to the nasopharynx or even to the tympanum has not been mentioned as a therapeutic possibility, and its use might prove of some value if the otologist has reason to believe that persistent tubal obstruction or tympanic hemangioma may be a causative factor. If there is X-ray evidence of mastoid pathology, it would seem that resort to simple cortical mastoidectomy could not be open to serious criticism. Certainly an attitude of expectant optimism in the face of recurrent hemotympanum with severe conductive deafness is to be deplored. Ranger's hypothesis that the cellular mastoid acts as a reservoir if not a source of the fluid suggests that obliteration of this cellular space is a logical procedure when more conservative measures fail. The outcome of his two cases apparently confirms this hypothesis so far as can be concluded from so few examples. Hoople's[9] experience with serous otitis media might be taken as at least analogous support. The mystery of serous effusion into the middle ear has been by no means solved and Hoople advocates resort to simple mastoidectomy in cases which resist all conservative efforts. He, too, feels that the cellular mastoid is at least a reservoir for the accumulation of fluid.

CASE REPORT.

Presentation of personal experience with another case of idiopathic hemotympanum may serve only to augment the meager reports thus far in the medical literature. With the puzzling experience of published authors in mind, this writer approached his own experience with a case, hopeful for some clue to the mystery. Thus far, the course of events has been amazingly parallel to those described by Ranger,[15] and no startling revelations have ensued.

This boy was first seen in March, 1947, at the age of eight years. He had had a tonsillectomy and adenoidectomy performed in another city one month previously and had made an apparently uneventful recovery. Four days before we saw him, he suddenly developed an earache on the left side, followed within a few hours by spontaneous otorrhea. He was

admitted to the hospital with an oral temperature of 100°, a slightly tender left mastoid, and a spontaneous perforation of the tympanic membrane in the antero-inferior quadrant. His recovery on adequate penicillin therapy was rapid and uneventful, and he was discharged from the hospital on the ninth day with the ear dry and the drum healed. No note was made at that time regarding any unusual color of the membrana tympani.

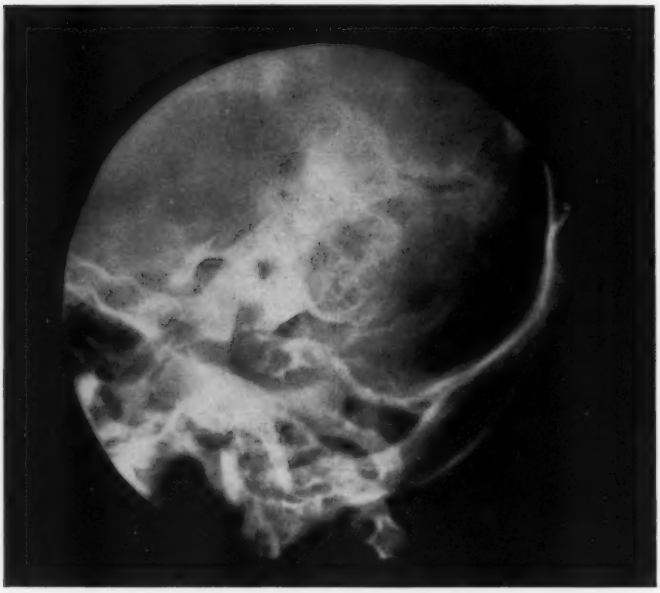

Fig. 1. Roentgenogram, left mastoid, June 19, 1952.

This boy returned to our office for checkup in May and again in August, 1947, complaining of no discomfort and apparently entirely well. His general ear, nose, and throat examination was entirely negative. Sinus X-rays were not taken, but transillumination was clear and his nose clean. There was no sign of residual tonsil or adenoid tissue, and examination of the nasopharynx with retropharyngeal mirror and nasopharyngoscope was negative.

The middle ear was easily inflated by the Politzer method and there was no clinical evidence of tubal obstruction. The Weber test, however, lateralized to the left, and the Rinne test indicated bone conduction to be much better than air conduction. At all times, the right ear remained entirely normal. An audiogram confirmed the normal findings in the right ear and revealed an almost flat loss of thirty-five decibels across the entire frequency range in the left ear. Bone conduction tests indi-

cated that this loss was strictly conductive. Again, no note was made to imply that the color of the left eardrum was unusual, only that the drum was intact. Inflation did not subjectively improve the hearing, and, inasmuch as there was no clinical evidence of tubal obstruction, this procedure was not repeated for therapeutic purposes.

In March, 1948, the patient returned for a routine checkup, and an audiogram showed the identical thirty-five decibel conductive loss observed seven months previously. Inflation was easily accomplished and this time elicited a favorable response, although the functional tuning fork tests continued to indicate a persistent conductive lesion. Now, for the first time, an unusual appearance of the left eardrum was noted. "The drum has a peculiar bluish appearance as of an unresolved blood clot". It would seem that we accepted this phenomenon without further comment and probably assumed that the hemotympanum would undergo spontaneous resolution.

We next observed the patient in September, 1948, because of a sudden spontaneous drainage from the same ear. No pain was felt, but there was evidence of a mild upper respiratory infection. His oral temperature was 99.6° but no systemic illness was evident and no mastoid tenderness. The drainage was purulent in character, and the spontaneous perforation was antero-inferior in location. He responded slowly to penicillin and sulfadiazine. The drum resumed its translucency, and the discharge lost its purulent character but became thick and brownish-red in color. Ten days after therapy was begun, this peculiar discharge was unabated, and again the unusual blue color of the drum was noted. This time, there was definite bulging of the posterior half of the drum and a generous myringotomy was performed. We were impressed by the relative insensitivity of the membrane and its loose, flaccid consistency. Drainage of the viscid fluid from the middle ear was promoted on several occasions by Politzer inflation, suction with the Siegle otoscope, and point suction. Within two weeks the ear was dry, and the drum in normal position, but again a dramatic dark-blue color. An audiogram in February, 1949, showed no change in the thirty-five decibel conductive loss in the left ear.

In March, 1949, following a mild upper respiratory infection and a brief earache, the left ear was again discharging the same brownish viscid material. At least five cubic centimeters of discharge were removed by suction and inflation, and the boy was given penicillin. The ear promptly healed and resumed its blue color. At no time was he systemically ill during this episode.

The patient was not seen again until July, 1950, when another identical episode occurred and again promptly subsided with institution of penicillin, inflations, and suction. On this occasion, mastoid X-rays were taken despite the apparently benign and indolent course of the disease. "Comparative stereoscopic studies of the mastoids and a special study of the petrous ridges show a well-developed right mastoid with a large number of air-containing cells. The right mastoid is normal. The development of the left mastoid is similar to that of the right, but there is diffusely increased density of the left mastoid. Trabecular structure is somewhat fuzzy and definitely altered from the normal in the antral area but appears to be intact in the tip, and over the sinus; but the plate of the knee of the sinus is not visible. No abscess formation or cholesteatoma is seen. *IMPRESSION:* "Normal right mastoid. Changes in the left mastoid are undoubtedly due to mastoiditis with an accumulation of fluid or granulation tissue, and there has been a destruction of the plate at the knee of the sinus. (signed) Russell Gates, M. D., Roentgenologist." By this time we finally realized that we were confronted by an unusual situation

and urged that a simple mastoidectomy be performed before another acute episode should occur. Because of a family complication beyond our control, we were obliged to temporize, with the understanding that surgery must be permitted as a possible emergency in the event of sudden evidence of intra-cranial complications.

Nothing further occurred until June, 1952. The boy had been swimming at frequent intervals during the Spring, and had developed a mild upper respiratory infection. Without pain or other warning symptoms, otorrhea suddenly recurred, and the entire clinical picture assumed the exact appearance of his previous episodes. Again the perforation healed in response to antibiotics and evacuation of the tympanum, but the eardrum retained its usual indigo hue and the hearing remained at thirty-five decibels below normal. Comparison X-rays by the same roentgenologist showed mastoid involvement identical with the findings of his previous films two years before. There was no visible change in the appearance of the blurred left mastoid cells and the apparent bone destruction over the knee of the lateral sinus. Further equivocation seemed futile and a simple mastoidectomy was finally performed on July 17, 1952.

Surgical exploration revealed an extensively pneumatized left mastoid structure with no visible sign of active infection. The cell septa were found to be intact and there were neither dural nor sinus exposures at any point. The radiological appearance of bone destruction in the region of the knee of the lateral sinus could readily be explained by the presence of extremely large pneumatic cells which were almost certainly physiological and not the result of necrosis. Every single mastoid cell from the additus ad antrum to the most remote post-sinus cell in the region of the mastoid emissary vein was found to be completely full of thick, reddish-brown material in which could be seen yellowish slimy crystals which tended to float on the surface of the underlying liquid. Both the additus and the antrum appeared to be abnormally large as if there might have been bone absorption from pressure distention. All cells were exenterated and a large myringotomy done. The post-auricular incision was closed after insertion of a number fourteen (French) fenestrated rubber tube into the mastoid antrum. It is possible that polyethylene tubing might have been superior for the purpose. This tube was permitted exit through the inferior angle of the operative wound.

During the patient's hospitalization various laboratory procedures were performed, their choice being dictated mostly by the reported experiences of the reviewed authors. Cultures of the liquid obtained during surgical exploration were negative for bacterial growth at the end of five days. The blood hemoglobin was 13.5 grams, 89 per cent, red cell count 4.78 million, white cell count 6,250, with 53 per cent segmented polymorphonuclear neutrophiles, 8 per cent immature stab forms, 28 per cent lymphocytes, 7 per cent mononuclears, 4 per cent eosinophiles, and 1 per cent basophiles. Coagulation time was four minutes, and bleeding time (Duke's method) was two minutes. Platelet count was 230,000. Prothrombin concentration was 70 per cent (normal 30 per cent to 100 per cent) and serum cholesterol 215 (normal 110 to 390). Routine and microscopic urinalysis was negative. The pathologist's report of microscopic findings follows: "a. Osteitis of cancellous bone; hemosiderin-containing macrophages and young fibroblastic proliferation. b. Chronic granulation tissue with foreign body giant cells, vessicular spaces from which cholesterol has been dissolved; old and recent hemorrhage. (signed) E. L. Benjamin, M. D., Pathologist."

The patient's post-operative course was smooth and afebrile. He was ambulatory after the first day and volunteered that his hearing had sub-

jectively improved almost immediately upon his return to consciousness after surgery. From the first, the sero-sanguinous drainage from both the myringotomy and the post-auricular tube was unusually profuse. At no time were there gross purulent characteristics to the fluid. All sutures were removed on the fourth post-operative day. He was discharged from the hospital on the sixth day and continued to be observed daily in our office. In order to avoid possible complications from intercurrent infection, he was maintained on a moderate daily dosage of intramuscular penicillin-streptomycin combination for another week. The tympanic membrane had healed by the tenth post-operative day; but the post-auricular drainage continued, and there was fluid visible in the middle ear. This fluid was easily ejected through the post-auricular tube by Politzer inflation which was performed daily to evacuate the middle ear. Following each evacuation of the tympanum, the eardrum was seen to resume a normal translucent gray color, only to have the blue color partially recur within twenty-four hours. Each day, the quantity of fluid removed decreased appreciably, and on the twenty-first post-operative day the drain was removed and the patient sent home to a nearby city.

After a two-week interval, the patient was rechecked and the post-auricular wound found to be completely healed. The eardrum was now in a normal position with no appearance of flaccidity or bulging, but the blue color was again visible in the inferior half of the drum. Inflation dispersed the blue color immediately and the drum was not incised. An audiogram done at this time showed a conductive hearing loss in the left ear of less than fifteen decibels, an improvement of approximately twenty decibels from the hearing prior to surgery.

Our most recent recorded observation on this patient was three weeks later. There were no subjective complaints, and the boy had been physically active, including vigorous sports and swimming. He stated that the ears felt "just alike" so far as he could discern; however, the left drum was again a dark blue color, although position and luster of the membrane were normal. A small inferior marginal myringotomy was done, and the middle ear easily cleared by Politzer inflation and suction in the canal. In spite of ample time for a large quantity of fluid to have formed, only about one-half cubic centimeter was obtained, and the drum resumed a normal color. The fluid obtained on this last occasion was no longer viscid, but of practically aqueous consistency and could have been easily aspirated with a twenty-four gauge needle. The color of the fluid, however, was still the characteristic chocolate or reddish-brown. It was fascinating to watch the blue drum change to pearly gray as the fluid was removed by suction. This observation satisfied our previous impression that the blue color is the result of brownish fluid seen through the translucent drum, rather than any color inherent in the membrane itself. At this time, we feel that we have accomplished removal of the fluid-reservoir, but not the point-source of the recurrent hemotympanum. It is our hope and belief that with the storage of large quantities of old blood obviated by exenteration of the mastoid structure, the massive accumulation will be prevented and the point-source of bleeding may subside. It is further our intention, if several subsequent evacuations of this tympanum fail to provide permanent relief, to consider X-ray treatment of the middle ear. Certainly, the boy's hearing loss is now so little as practically to prohibit a radical mastoid procedure. Even a modified radical might well fail to reveal any source of bleeding and could be expected to have a possible adverse effect on his now serviceable hearing. Our future conduct will be guided by the patient's response to the present conservative regimen.

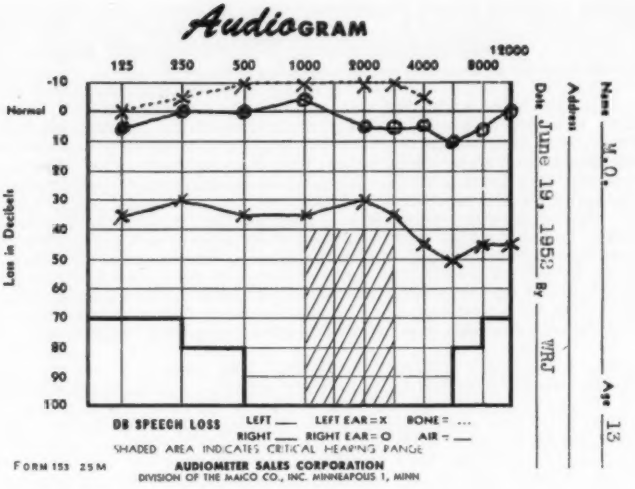

Fig. 2. Before surgery.

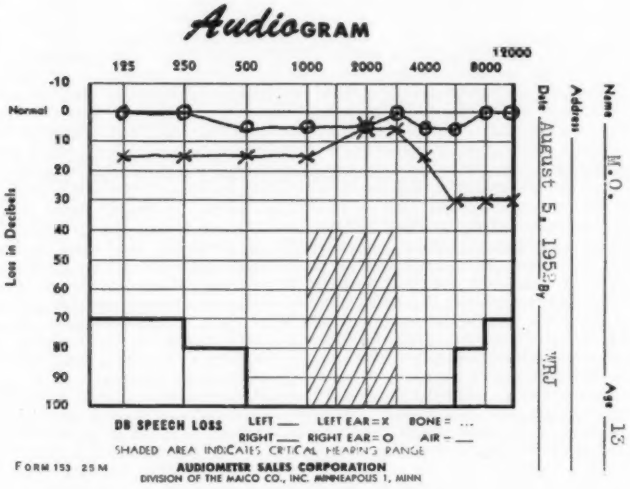

Fig. 3. After surgery.

Since the beginning of our experience with the above case, we have had the opportunity to observe an identical condition in an eight-year-old Chinese girl. Unfortunately we were confronted by the obstacle of ancient Oriental prejudice on the part of the child's guardian and no definite action could be taken thus far. Tonsillectomy and adenoidectomy, repeated inflations, and several evacuations of the tympanum by myringotomy have failed to prevent re-accumulation of fluid and re-appearance of the blue eardrum. In this child the hearing loss is at a fifteen decibel level, and because of the absence of alarming signs or symptoms, the family has been unwilling to send her in for consistent observation or even X-ray studies. Perhaps it will be our fortune to observe this case more adequately at some future time, inasmuch as there is no reason to believe that her hemotympanum will resolve spontaneously.

SUMMARY.

The extant literature pertaining to hemotympanum has been reviewed and, so far as possible, correlated. The reports of hemotympanum from known causes have been separated from those of unknown etiology, and this latter idiopathic group of ten cases studied for comparison. Most authors have considered the condition to be a rare, perplexing oddity, whose origin is obscure and whose treatment is subject to conjecture and dispute. Of the ten cases designated as a disease entity, only two have been explored surgically after exhaustive clinical study. Opinions of various authors regarding the cause for this uncommon disease include: *1.* rupture of a hemorrhagic bleb in acute otitis media; *2.* protrusion of the jugular bulb through a dehiscence in the hypotympanum; *3.* chronic Eustachian tube obstruction; and *4.* recurrent hemorrhages into the so-called middle ear cleft from an undetermined site in the mastoid, middle ear, or Eustachian tube. These last-mentioned recurrent hemorrhages are of admittedly unknown etiology, and even their exact point-source has not as yet been determined.

Authors agree remarkably well in regard to the clinical appearance of the blue eardrum, the conductive loss of hearing, and the persistent tendency of fluid to re-accumulate in the middle-ear cleft. Their descriptions of the fluid obtained by myringotomy are in perfect accord, but there is as yet no consensus of opinion regarding definitive treatment. Two of the reported cases subsided after tonsillectomy and adenoidectomy. A third case obtained partial relief from

myringotomy and inflations, and three others were unaltered by this system of treatment. One case was merely reported as a medical curiosity with no therapy described, and another was assumed by the otologist to be the manifestation of a venous anomaly and rather indefinite treatment instituted. Of the two cases surgically explored, one obtained improved hearing, but both recovered from the hemotympanum. Compilation of opinions regarding a consistent therapeutic regimen indicates that if elimination of concurrent nose and throat disease and nasopharyngeal lymphoid tissue, and repeated myringotomy and inflation with thorough evacuation of the middle ear contents, fail to alleviate the condition, and mastoid X-rays show pathology, simple mastoidectomy is indicated.

The writer presents a case in detail which had been followed conservatively for five years, and finally explored surgically after lesser measures gave no lasting satisfaction. From the standpoint of time, this case is necessarily not complete, inasmuch as there has been a tendency for fluid in small amounts to recur, and insufficient time has elapsed to determine if this accumulation will cease after several evacuations. The hearing has been markedly improved, and the clinical results thus far have seemed to justify the surgery performed. No additional information has been obtained in spite of reasonably complete laboratory, X-ray, and pathology studies, which will satisfactorily explain the cause or exact source of the recurrent hemorrhage. The cause of the diagnostic blue color of the eardrum has been demonstrated to be the collection of degenerated blood constituents as visualized through the translucent membrana tympani. The importance of maintaining post-auricular drainage for at least three weeks following surgery is pointed out, but recognition of the uncertainty of complete cure is frankly admitted.

The writer also cites a second case, the thorough investigation of which was not possible, but in which the clinical appearance and course allied it to the subect under discussion.

CONCLUSIONS.

The etiology of the syndrome of the blue eardrum would seem to be as yet in the field of philosophy and speculation. There are many aspects of the condition which suggest its possible relation to the condition known as serous otitis media. Hoople[8] describes variations in the color of the exudate depending on the amount of microsocopic blood present. The analogy can be extended to include a comparison of the persistence and annoying recurrence of the two conditions in spite of almost any known therapy. Finally, we note that almost in desperation, mastoidectomy has been the ultimate treatment for selected cases in both categories.

Eagle's[4] scholarly discussion of serous otitis media implies the same element of mystery. He comments upon a vague effect of temperature and humidity changes upon serous exudates in the middle ear and points out that the condition is not infrequently bilateral. At once it can be observed that here may be an important point of disparity. Bilateral idiopathic hemotympanum has never been reported in the literature. This would tend to suggest that the latter disease is of local rather than systemic origin and probably not the result of any exogenous agent acting upon the organism as a whole.

If hemotympanum of traumatic origin is dissipated by absorption, why should the idiopathic variety seem to defy the usual course of events? Eggston and Wolff[5] refer to Robison[16] in their excellent description of the lymphatics of the nasal structures. They believe that lymphatic flow from the middle ear and Eustachian tube join the lymphatics from the nose and accessory sinuses in the pre-tubal lymph nodes. Normal flow of lymph then passes into the retropharyngeal and finally the jugular chain of lymphatics. They further maintain that any obstruction along this pathway results in retrograde lymphatic flow, which may explain middle ear infections as a complication of a homolateral sinusitis. Anastomosis with the contralateral lymphatic chain is asserted to be across the soft palate and possibly to a limited extent across the retropharyngeal space. It is not impossible that

some lymphatic obstruction might account for delayed absorption of hemorrhage in the middle ear, but it assuredly fails to explain the source of recurrent bleeding.

Admitting the actual occurrence of dehiscences in the hypotympanum, and recognizing that the jugular bulb can project into the middle ear, we are doubtful if any such condition is responsible for the entity we refer to as idiopathic hemotympanum. Why should a jugular vein or its tributaries repeatedly leak into the middle ear without the slightest effort of nature to repair the supposed defect in the wall of the vein? If bulb protrusion is the cause of blue eardrum, why have repeated myringotomies upon the dozen or so reported cases not resulted in obvious penetration of the jugular bulb? As a matter of fact, the myringotomies in our own cases were frequently done in the border of the central inferior quadrant.

By a process of elimination, we have arrived at the conclusion that somewhere in the middle ear cleft of these patients, exists an area af varicosity or even hemangioma which fails to undergo complete tissue repair but repeatedly breaks down to permit the escape of fresh blood. Apparently Ranger[15] was able to destroy such an area when he exenterated the mastoid, but if such an area were located in the tympanum or within the Eustachian tube, mastoidectomy would serve only to remove a reservoir full of old blood; hence the inclination to try irradiation in our own case if the condition persists.

Since, to the best of our knowledge, only three cases of idiopathic hemotympanum have been surgically explored, no clear-cut regimen of treatment can be exploited. Obviously conservative methods must be attempted until their efficacy is proven or disproven. We believe that if more otologists will be alert to detect this supposedly rare condition and will pursue such cases to ultimate surgery if necessary, the answer to the riddle will be forthcoming. Some day, one of us will exercise the temerity of performing a complete radical mastoid operation upon one of these cases and perhaps find the true explanation.

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TOPICAL THERAPY OF DISTURBANCES OF THE UPPER RESPIRATORY TRACT.

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In most instances acute rhinitis terminates within a few days after onset without benefit of special treatment. It is, therefore, difficult to evaluate either systemic or topical therapy in acute rhinitis because of this tendency for spontaneous cure. On the other hand, rhinitis which persists for seven days often continues for a longer period and not infrequently is associated with complications if proper therapy is not given.[1,2] Many such complications are bacterial in origin, often due to streptococci and at other times to pneumococci, *Hemophilus influenzae*, etc., and some are due to allergy. Although successful results are more difficult to obtain in the persistent cases, clinical evaluation of the therapeutic potential of a preparation is more likely to be significant. The following study of the therapy of rhinitis and sinusitis was, therefore, limited to such cases.

The ideal topical preparation for the nasal mucosa should relieve "stuffiness" promptly and for prolonged periods without toxicity. It should have a pH below 7.0, be isotonic and buffered and contain no ingredients which inhibit ciliary activity.[3] Infants and children particularly often fail to take adequate fluids or food because an obstructed nares makes swallowing difficult or uncomfortable. A good preparation should relieve this and facilitate nursing. Shrinking of the nasal mucosa may further aid to drain sinuses by helping the ostia to remain open.

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An ideal preparation should not intensify the symptoms and should not irritate the mucous membrane, especially if used for longer than four or five days in succession; it should not contain oil. There should be minimum absorption of the effective constituents and minimum incidence of sensitivity reaction.

Ideally one might choose the ingredients for a topical nose drop or spray on the basis of the specific etiology of the condition; however, it is not practical to obtain reports of cultures and often even of smears and other procedures during the first examination. It is sometimes difficult to make a differential diagnosis on clinical evidence between allergic rhinitis, bacterial rhinitis or sinusitis, and similar upper respiratory tract disturbances due to other etiologic factors; therefore, a preparation relatively free from untoward reactions and which would provide effective therapy against the majority of etiologic factors in upper respiratory disease is desirable.

The preparation* chosen for this study appeared to conform to these requirements. It contains phenylephrine hydrochloride in a concentration of 0.25 per cent as the vasoconstrictor or decongestant agent. This is a widely used, powerful and safe agent. Its use is associated with minimal re-turgescence and little or no central or cardiac stimulating effect.[4,5]

The antihistamine used in this preparation is thonzylamine hydrochloride in a 1 per cent solution. This agent was found by Criep and Aaron[6] to provide prompt symptomatic relief in allergic rhinitis.

Among the more common bacteria found in nasal infections are beta hemolytic streptococci, pneumococci and hemolytic *Staphylococcus aureus*. Gramicidin, which is produced by *Bacillus brevis*, has a high degree of *in vitro* bactericidal activity against these bacteria.[7] Dubos showed that experimental animals may be protected against infection with pathogenic organisms when gramicidin is applied locally.[8] It has the same order of activity against strains of Gram-positive

*Biomydrin was generously supplied by the Nepera Chemical Company, Inc., Nepera Park, Yonkers, New York.

cocci in tissue culture as a highly concentrated preparation of penicillin.[9] Gramicidin is present in the preparation used in a concentration of 5 ug/ml. This concentration in *in vitro* tests was sufficient to inhibit strains of hemolytic streptococci, pneumococci and staphylococci.[10]

Neomycin is also active against a wide variety of Gram-positive bacteria. Of greater importance is its effectiveness against many Gram negative bacteria such as *Hemophilus influenzae*.[11] Neomycin is derived from a strain of *Streptomyces fradiae*[12] and is present in this preparation in a concentration of 100 ug/ml.

The combination of neomycin and gramicidin for topical use has a unique advantage since these antibiotics are not given parenterally, and, therefore, possible sensitization is correspondingly less important. Since each is bactericidal, bacterial resistance is less likely to occur or is slow to develop.

Effective antibiotic, antihistamine or vasoconstrictor agents which cannot reach mucous membrane surfaces because of thick overlying mucus or purulent material naturally will be much less effective. Many substances are now available which because of their low surface tension tend to disperse and reduce the viscosity of these secretions. Thonzonium bromide is such a surface acting agent. It has the additional desirable property of being a potent bactericide itself and it may actually enhance the activity of the gramicidin-neomycin combination.[13] It is present in this preparation in a concentration of 0.05 per cent. The preparation is stable, has a pH of 6.2 and is isotonic and buffered.

Busis and Friedman[14] have already reported excellent results with this preparation. The present study was done for the most part before their report appeared.

The purpose of this study was twofold: to study the bacterial flora in upper respiratory disturbances and to evaluate this interesting and potentially valuable combination of agents. The latter evaluation was planned to determine both patient acceptability and clinical and bacteriological effectiveness.

The 50 patients on whom adequate follow-up could be made ranged in age from five months to 80 years. Twenty-two were children under 15 years of age. Most of them were patients in the senior author's otolaryngology clinic, or in his private office. Eleven were patients in the hospital; the others were ambulatory. They all had had rhinitis or nasal congestion, infection or other related complaint for at least seven days before therapy was begun. The patients, therefore, were a selected group composed of those who had failed to respond either to no therapy or to other forms of therapy. The physicians of the general clinic cooperated to extend this series by referring to the senior author's clinic all patients who had persistent rhinitis or sinusitis which had failed to respond to conventional therapy.

The clinical diagnosis was established after careful examination of each patient. The diagnosis of sinusitis was supported by evidence from transillumination or X-ray. Smears for eosinophiles were made whenever allergy was suspected. Cultures were obtained by the use of a cotton swab on a copper wire which was passed through the nose into the nasopharynx. In follow up studies at least four hours were allowed to elapse between the last use of the drug and the taking of the culture. Routine bacteriological methods were used to identify the predominating organisms (see Table I*).

The patients were urged to take "plenty of fluids," but no other medication was given. When much exudate was present, it was aspirated upon each visit, taking care to avoid trauma to the membrane. The topical preparation being studied was used to shrink the mucosa so that more adequate suction could be carried out. For this purpose, cotton pledgets were soaked in the solution and inserted into the nose and allowed to remain in place for three to five minutes.

Technique of self administration of the drug was taught each patient. If the child was too young to learn the technique, it was taught to the parent. For the infants and young children the Proetz position was used to instill drops. The

*In the table the presence of such ordinarily non-pathogenic organisms as **Staphylococcus albus** is not noted although some questionably pathogenic ones such as **E. coli** and **Aerobacter aerogenes** are included.

TABLE. I.

EVALUATION OF BIOMYDRIN IN UPPER RESPIRATORY DISTURBANCES.

Case No.	Age	Pre-treatment Clinical Diagnosis	Predominant Symptoms or Signs	Culture		Estimate of Clinical Result
				Pre-treatment	Post-treatment	
1	50 yrs.	Atrophic Rhinitis	Crusting, post nasal drip	Beta hem. Str.	no pathogen	Good
2	65 yrs.	Atrophic Rhinitis	Crusting and mucoid ? discharge	Ps. aeruginosa	same	Good
3	70 yrs.	Atrophic Rhinitis	Crusts and mucoid ? discharge	Beta hem. Str.	no pathogen	Good
4	71 yrs.	Atrophic Rhinitis and Chronic Sinusitis	Crusts, discharge and pus in antrum	Beta hem. Str. A. aerogenes**	A. aerogenes	Good
5	50 yrs.	Atrophic Rhinitis	Post nasal drip	Beta hem. Str.	no pathogen	Good
6	43 yrs.	Atrophic Rhinitis	Crusting	Beta hem. Str.	no pathogen	Good
7	47 yrs.	Atrophic Rhinitis	Crusting, boggy mucosa	H. influenza A. aerogenes Hem. E. Coli	A. aerogenes	Good
8	43 yrs.	Atrophic Rhinitis	Crusting	Beta hem. Str. Staph. aur.***	no pathogen	Good
9	60 yrs.	Atrophic Rhinitis	Crusting	Beta hem. Str.	Beta hem. Str.	Good
10	29 yrs.	Allergic Rhinitis	Sneezing, watery discharge with obstruction	Hem. E. coli	same	Only temporary symptomatic relief
11	22 yrs.	Allergic Rhinitis	Pale mucosa, watery discharge, eosinophiles	Staph. aur.	same	Relief only with packing
12	7 yrs.	Allergic Rhinitis	Pale mucosa, watery discharge, eosinophiles	Ps. aeruginosa†	same	Fair
13	23 yrs.	Allergic Rhinitis	Post nasal drip	No pathogen	same	Good

Case No.	Age	Pre-treatment Clinical Diagnosis	Predominant Symptoms or Signs	Culture Pre-treatment	Culture Post-treatment	Estimate of Clinical Result
14	2½ yrs.	Allergic Rhinitis	Boggy mucosa	Staph. aur.	same	Fair
15	42 yrs.	Allergic Rhinitis	Boggy, pale mucosa	Hem. E. coli A. aerogenes	same	Fair
16	6 yrs.	Allergic Rhinitis	Post nasal drip, obstruction	P. mirabilis†† Ps. aeruginosa	same	Fair
17	47 yrs.	Allergic Rhinitis	Post nasal drip	No pathogen	same	None
18	10 yrs.	Allergic Rhinitis	Very pale mucosa. Polypi.	A. aerogenes	same	Good
19	17 yrs.	Allergic Rhinitis	Pale mucosa	Staph. aur.	--	Patient developed "grippe" 3 days after Rx started
20	11 yrs.	Allergic Rhinitis	Sneezing, watery discharge. Obstruction	Staph. aur.	No pathogen	Fair
21	10 yrs.	Allergic Rhinitis Acute Sinusitis	Perennial and Acute	A. aerogenes Staph. aureus	--	Good to fair with oft repeated Rx
22	6 yrs.	Allergic Rhinitis	Sneezing, obstruction	Staph. aureus	same	Fair
23	9 yrs.	Allergic Rhinitis	Pale mucosa, eosinophiles	B. subtilis	--	None
		Allergic Rhinitis Superimposed Infection	Same with purulent drainage	Pneumococci Group A, type 7 Staph. aureus	No pathogen	Good for superimposed infection
24	47 yrs.	Allergic Rhinitis Otitis Media, catarrhal	Nasal congestion. Mucoid otorrhea	Staph. aureus (pure culture)	same	None

TABLE I. (Continued)
EVALUATION OF BIOMYDRIN IN UPPER RESPIRATORY DISTURBANCES.

Case No.	Age	Pre-treatment Clinical Diagnosis	Predominant Symptoms or Signs	Culture		Estimate of Clinical Results
				Pre-treatment	Post-treatment	
25	9 yrs.	Allergic ? Rhinitis	Pale, boggy mucosa	No pathogen	--	Fair temporary symptomatic relief
26	5 yrs.	Allergic Rhinitis	Sneezing, watery discharge. Obstruction	--	--	Temporary relief only
27	12 yrs.	Allergic Rhinitis Acute Sinusitis	Pale mucous membranes. Cloudy antrum	No pathogen	No pathogen	Good
28	6 yrs.	Allergic Rhinitis	Severe sneezing and thin discharge	No pathogen	--	Good temporary relief
29	35 yrs.	Subacute Rhinitis Otitis Media	Nasal congestion Vesicles on ear drum	Ps. aeruginosa	same	Fair
30	55 yrs.	Chronic Rhinitis	Obstruction Purulent discharge	Ps. aeruginosa Staph. aur.	same	None
31	73 yrs.	Chronic Rhinitis	Post nasal drip	Ps. aeruginosa Staph. aur.	--	Fair
32	3 yrs.	Chronic Rhinitis	Obstruction Purulent discharge	Staph. aur. A. aerogenes	A. aerogenes	Good
33	37 yrs.	Chronic Rhinitis Rt. Maxillary Sinusitis	Deviated septum, purulent discharge, yellow to brown. Diabetes mellitus	Staph. aur.	same	During Rx discharge cleared but afterward returned. Medication "irritated" nose

Case No.	Age	Pre-treatment Clinical Diagnosis	Predominant Symptoms or Signs	Culture Pre-treatment	Culture Post-treatment	Estimate of Clinical Result
34	44 yrs.	Chronic Rhinitis	Red mucous membranes. Post nasal drip	A. aerogenes	--	Good
35	7 yrs.	Chronic Rhinitis	Inflamed nasal mucosa, purulent discharge	Pneumococcus type undetermined	No pathogen	Good
36	5 mos.	Chronic Rhinitis	Profuse mucopurulent discharge	Staph. aur.	same	Good
37	13 yrs.	Chronic Rhinitis Acute Maxillary Sinusitis	Purulent discharge Obstruction, left antrum	Staph. aur.	same	Improved while receiving drug, but recurred when drug was stopped
38	3½ yrs.	Chronic Rhinitis	Yellow purulent discharge	Staph. aur.	No pathogen	Good
39	4½ yrs.	Chronic Rhinitis	Red mucous membranes Post nasal discharge	No pathogen	--	Good
40	8 yrs.	Chronic Rhinitis	Thin purulent discharge	Beta hem. Str.	No pathogen	Good
41	6 mos.	Chronic Rhinitis Otitis Media, catarrhal	Purulent discharge	H. influenza	No pathogen	Good
42	32 yrs.	Acute Maxillary Sinusitis	Obstructed antral sinus Purulent discharge	Beta hem. Str.	same	Good
43	29 yrs.	Acute Maxillary Sinusitis	Pain Purulent discharge	E. coli A. aerogenes	--	Good
44	48 yrs.	Subacute Sinusitis	Obstruction Purulent discharge	Beta hem. Str.	No pathogen	Good

TABLE I. (Continued)
EVALUATION OF BIOMYDRIN IN UPPER RESPIRATORY DISTURBANCES.

Case No.	Age	Pre-treatment Clinical Diagnosis	Predominant Symptoms or Signs	Culture Pre-treatment	Culture Post-treatment	Estimate of Clinical Result
45	43 yrs.	Acute Maxillary Sinusitis	Purulent discharge	E. coli A. aerogenes	--	Good
46	69 yrs.	Chronic Maxillary Laryngitis Atrophic Rhinitis	Laryngitis sicca of years duration, thick nasal discharge Hemachromatosis	H. influenza	No pathogen	Good while receiving drug. Recurrence upon stopping it.
47	12 yrs.	Chronic Sinusitis Acute Exacerbation	Boggy mucous membrane Cloudy sinus	P. vulgaris†††	No pathogen	Good
48	10 yrs.	Chronic Sinusitis Acute Exacerbation	Pain, headache Purulent discharge	E. coli	--	Fair
49	15 yrs.	Subacute Sinusitis	Headache. Cloudy maxillary antrum. Red mucous membrane	Beta hem. Str.	No pathogen	Good
50	80 yrs.	Vasomotor Rhinitis	Watery discharge Multiple polyps	No pathogens	--	Temporary

- - no data
*Beta hem. Str. = Beta hemolytic streptococci
**A. aerogenes = Aerobacter aerogenes
***Staph. aur. = Hemolytic Staphylococcus aureus, coagulase positive
†Ps. aeruginosa = Pseudomonas aeruginosa
††P. mirabilis = Proteus mirabilis
†††P. vulgaris = Proteus vulgaris

head was extended so that the chin and external auditory meatus were in a vertical plane, and the drug was then instilled in the form of drops. The head was allowed to remain in this position for about one minute. Sinus displacement was not the purpose of this technique nor was it accomplished thereby. In all other cases, the preparation was introduced by spraying from a special squeeze bottle, into each nostril, with the patient sitting upright. The patient was then instructed to sniff a few times. Infants under two years of age were given one to three drops in each nostril three times daily. Children two to 15 years were given one to two sprays in each nostril, and adults, two to three sprays four times daily. When nostril obstruction interfered with feeding, the preparation was instilled 15 to 20 minutes prior to feedings. It is interesting to note that older children and adults generally preferred to use the special squeeze bottle as a spray rather than instill the drug as drops.

Treatment with this preparation was prescribed for from three to 14 days depending upon the condition. The patients were given the drug in a special plastic atomizer of the "squeeze bottle" type. Follow-up studies were made within one week after starting therapy and as frequently thereafter as indicated. Clinical response was judged as Good, Fair, Temporary or None (see Table I). This was an estimate based upon the judgment of the investigators with due consideration also for the opinion of the patient. Where response could not be so classified, there is a self-explanatory statement in the table.

RESULTS.

In nine of the patients, the initial clinical diagnosis was atrophic rhinitis. These patients varied in age from 43 years to 71 years. The authors are in agreement with the current opinion that atrophic rhinitis is not primarily due to infection. It is, therefore, of more than passing interest that cultures of beta hemolytic streptococci were obtained in seven of the nine cases and H. influenza in one of the two remaining cases. The presence of such pathogens might be expected to aggravate this condition seriously. After topical use of this

preparation, beta hemolytic streptococci were cultured from only one of the seven patients. This is probably the reason for the good clinical response in all of these cases.

In 19 of the patients, the initial clinical diagnosis was allergic rhinitis. The age ranged from five to 47 years. In six, no pathogens were found on culture. One case had an obvious superimposed infection due to *Pneumoccocus*, type 7. It is difficult to evaluate the etiologic significance of the predominant organism in the remaining cases. Seven showed hemolytic *Staphylococcus aureus*, coagulase positive, two hemolytic *E. Coli*, two *Pseudomonas aeruginosa*, and one *Aerobacter aerogenes*. Some of these may have emerged as the result of overgrowth following previous administration of other antibiotics and may have had little effect on the current symptoms or signs. In four of the 19 patients relief from symptoms and signs was classified as good, and the improvement persisted even after the medication was stopped. It is tempting to postulate that in these cases bacterial invasion had complicated or aggravated the allergic symptoms and that the relatively prolonged remissions following cessation of therapy in some cases could be attributed to the bactericidal properties of the medication. In two there was no response. In the majority (11 cases), however, improvement was most notable while the medication was being given, and symptoms returned after the medication was stopped; a finding in accord with the allergic etiology in these cases. One of these patients developed "grippe" three days after the medication was started. In one there was a superimposed rhinitis with purulent drainage from which *Pneumococcus*, type 7, was cultured. This complication cleared up rapidly with the use of this preparation.

One patient with severe vasomotor rhinitis and nasal polyps experienced brief temporary relief while receiving the medication.

Thirteen patients had either subacute or chronic rhinitis often with purulent discharge. The ages ranged from five months to 73 years. A variety of possibly pathogenic organisms were cultured; *Staphylococcus aureus* in four; *Staphy-*

lococcus aureus plus *Pseudomonas* in one; *Staphylococcus aureus* plus *Aerobacter aerogenes* in one; *A. aerogenes* alone in one; *Pseudomonas* alone in one; *Pneumococcus,* type undetermined in one; beta hemolytic streptococcus in one and *H. influenzae* in one. The clinical response was good in eight, fair in two, temporary in two, and in one there was no apparent response.

In eight of the patients, acute or chronic sinusitis was the major problem. The age ranged from 10 to 69 years. Again a variety of organisms predominated, some of which were only questionably pathogenic. In two beta hemolytic streptococci were found; two showed both *E. coli* and *A. aerogenes;* one *A. aerogenes* alone; one *E. coli* alone; one *B. proteus,* one *H. influenzae.* In five, the clinical response was good, in one fair, and in two improvement was temporary.

If all the patients who had these upper respiratory difficulties are grouped together, the clinical response can be summarized as follows: Good in 26, or 52 per cent; good but lasting only while drug was being administered in 16 or 32 per cent; fair in three, or 6 per cent. In only three, or 6 per cent did the drug fail to produce any improvement. In one case the superimposed infection responded well, but the allergic rhinitis was relieved only while the drug was administered. In one the patient developed "grippe" while receiving the drug.

The effects of treatment on the bacterial flora of the nose are shown in Table I. Only Gram positive potential pathogens were found in 22 of the patients prior to treatment and these were not found on culture taken after treatment from 12 of these patients (or 54 per cent). At least one of these Gram positive organisms persisted after the treatment of nine, or 41 per cent of such cases. Gram negative invaders tended to persist more often after treatment. Of 16 patients found to harbor Gram negative organisms before treatment, only three (19 per cent) failed to reveal the same organism on culture after treatment. Of five patients who harbored mixed cultures of Gram positive and Gram negative organisms before treat-

ment, post-treatment cultures revealed no Gram positive organisms, but in each case the Gram negative organisms were still present.

The bacteriologic results may be summarized by noting that of 43 patients whose nasal cultures revealed one or more organisms of potential or possible pathogenicity before treatment, cultures after three to 14 days of treatment revealed no pathogen in 15 or 35 per cent. In three additional patients whose cultures contained two or more suspected pathogens before treatment, at least one of the organisms previously present was not found following treatment. Thus following treatment previously present potential pathogens were not found in 42 per cent of the patients so examined.

The clinical results in general corresponded to the bacteriologic findings in all the conditions studied including atrophic rhinitis, allergic rhinitis, chronic rhinitis, and sinusitis. In view of these findings, it seems pertinent to consider that in those conditions assumed ordinarily not to be basically infectious in origin, the symptomatology may be complicated or aggravated by a superimposed infection. If such is the case, treatment aimed at eradication of the offending organism may contribute to improvement.

The older children and adults generally preferred the spray to the drops. Within a few minutes after the spray, it was easy to demonstrate marked decongestion of the nasal mucosa. The patients generally noted prompt relief of symptoms and often improved ventilation and drainage. When improvement occurred in the patients with allergic rhinitis, the duration of the improvement varied considerably from individual to individual. On the whole, however, it was prolonged enough to be "welcomed" by the patients.

The only untoward effect observed was in a 37-year-old patient with chronic rhinitis and maxillary sinusitis who complained that the drug "irritated" his nose. He spontaneously discontinued the drug four days after he had begun to use it. None of the patients showed any evidence of anosmia or

parosmia. The children particularly noted no burning or stinging. After their first experience with it, they generally accepted it without objection.

SUMMARY AND CONCLUSIONS.

The effectiveness of a new topical preparation (Biomydrin) on a variety of disturbances of the upper respiratory tract was evaluated in a selected group of patients. The patients studied included only those with persistent symptoms which had failed to respond either by spontaneous remission or to conventional therapy.

The treatment used was particularly effective in infections due to susceptible bacteria. The preparation used was also effective in relieving allergic symptoms. This action was rapid and relatively prolonged.

Good responses were obtained in five of eight patients with sinusitis; fair response in one, and temporary relief in two patients whose symptoms reappeared after treatment was discontinued. Similar good results were obtained in nine of 12 patients with chronic rhinitis.

Seven of the nine patients with atrophic rhinitis were found to harbor beta hemolytic streptococci. Although the significance of this finding is not clear, the use of this preparation not only resulted in good clinical response but also improvement appears to have been concurrent with elimination of these organisms from the nasal cultures.

This preparation is a safe, well tolerated and effective solution for the topical treatment of either atrophic, allergic or infectious disorders of the upper respiratory tract.

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HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

November 1, 1953.

Acousticon Models A-17 and A-185.

Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.

Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.

Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Symphonette; Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."

Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.

Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.

Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J; Maico Top Secret Model L; Maico Maxitone.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Classic Model T9; Microtone Model T10; Microtone Model T612; Microtone Model 45.
Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Model D (Duplex); National Ultrathin Model 504; National Vanity Model 506.
Manufacturer: National Hearing Aid Laboratories, 106 So. 7th St., Philadelphia 6, Pa.

Normatone Model C and Model D-53.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.
Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, F-2 and F-3; Otarion Model G-2; Otarion Model G-3; Otarion Model H-1; Custom "5."
Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model Y (YM, YC and YC-7) (Veri-Small).
Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).
Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.
Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model J-92; Silvertone Model P-15.
Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.
Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.
Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988 and Sonotone Model 1010.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.
Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.
Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED.

Maico Transist-Ear, Model O; 3 transistors and 1 battery.
Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis, 1.

Otarion Model C-15; 1 transistor, 2 tubes, and 2 batteries (A & B).
Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill.

Sonotone Model 1010; 1 transistor, 2 tubes, and 2 batteries (A & B).
Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes, and 2 batteries (A & B).
Manufacturer: Telex, Inc., Telex Park, St. Paul, 1.

Zenith Model Royal-T; 3 transistors, and 1 battery.
Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).
Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Western Section: Saturday, Feb. 6, 1954, Portland, Ore., University of Oregon Medical School.

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ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

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Vice-Presdente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

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Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
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Vocal: Dr. Pedro Hernández Gonzalo.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa. U. S. A.
Meeting: 3rd International Congress of Broncho-Esophagology.
Time and Place: September or October, 1954, Lisbon, Portugal.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER — CAMPINAS.

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First Secretary: Dr. Roberto Barbosa.
Second Secretary: Dr. Roberto Franco do Amaral.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho, Dr. Penido Burnier and Dr. Gabriel Porto.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

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Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Díaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asís R., Dr. Jorge Bergallo Yofre.

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Vice-Presidente: Dr. V. R. Carri.
Secretario: Dr. Renoto Segre.
Pro-Secretario: Dr. Carlos A. Gutierrez.
Tesorero: Dr. J. M. Tato.
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SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLOMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

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Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

THIRD LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Franze Conde Jahn, Caracas.
Secretary: Dr. Victorino Marquez Reveron, Caracas.
Meeting: Caracas, Venezuela, Feb. 21-25, 1954.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

The following is a schedule of 1954 Meetings:

Eastern Section	Fri., Jan. 8th	New York City	The Waldorf-Astoria
Council Meeting	Sat., Jan. 9th	New York City	The Waldorf-Astoria
Southern Section	Sat., Jan. 16th	Louisville, Ky.	Brown Hotel
Middle Section	Mon., Jan. 18th	St. Louis, Mo.	Park Plaza Hotel
Western Section	Sat., Feb. 16th	Portland, Ore.	University Oregon Medical School

(In Portland, room reservations may be made at the Heathman Hotels.)

ANNUAL MEETING, Tues.-Thurs., May 25-27
Boston, Mass., Hotel Statler

May we call your attention to some of the attractions of Boston: Scene of the "Boston Tea Party" and the Battle of Bunker Hill; site of Faneuil Hall, the "Cradle of Liberty"; and the Old North Church (from the steeple of which the signal was given to Paul Revere); residences of Henry Wadsworth Longfellow, Wendell Phillips and William Lloyd Garrison; a short ride to the battle fields of Concord and Lexington, and the homes of the Alcotts, Emerson, Hawthorne and Thoreau; the birthplace of the Christian Science and Unitarian movements; and for those more interested in matters at hand, Durgin-Park for excellent food in a public market atmosphere; also the Boston Pops Orchestra for enjoyable music.

Reservations at the Boston Statler may be made by addressing Mr. Fred W. Potts, Sales Manager of the hotel.

For further information contact C. Stewart Nash, M.D., Secretary, 708 Medical Arts Bldg., Rochester (7), N. Y.

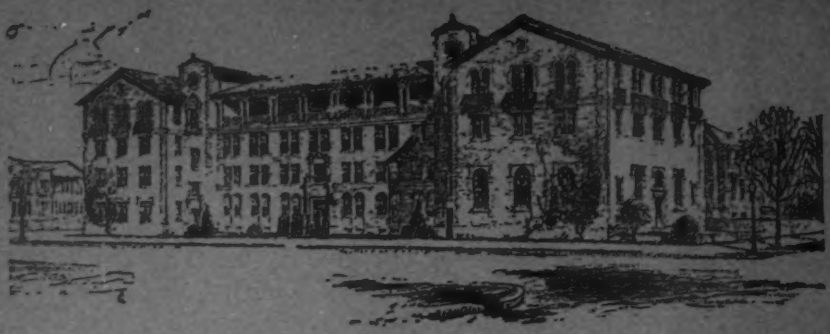
GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Georgia Society of Ophthalmology and Otolaryngology will hold its Spring meeting March 5th and 6th, 1954, at the General Oglethorpe Hotel, Savannah, Georgia. The speakers will be Dr. Paul Chandler, Boston; Dr. A. B. Reese, New York; Dr. Henry P. Wagener, Rochester; Dr. L. R. Boies, Minneapolis; Dr. Francis LeJeune, New Orleans, and Dr. J. H. Maxwell, Ann Arbor.

MIDWINTER SEMINAR IN OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Eighth Annual University of Florida Midwinter Seminar in Ophthalmology and Otolaryngology will be held at the Sans Souci Hotel, Miami Beach, the week of January 18, 1954. The lectures on Ophthalmology will be presented on January 18, 19 and 20, and those on Otolaryngology on January 21, 22 and 23. A midweek feature will be the Midwinter Convention of the Florida Society of Ophthalmology and Otolaryngology Wednesday afternoon, January 20, to which all registrants are invited. The registrants and their wives may also attend the informal banquet at 8 p.m., on Wednesday. The Seminar schedule permits ample time for recreation.

The Seminar lecturers on Ophthalmology this year are: Dr. W. B. Anderson, Durham, N. C.; Dr. W. P. Beetham, Boston; Dr. W. C. Owens, Baltimore; Dr. A. B. Reese and Dr. M. C. Wheeler, both of New York City. Those lecturing on Otolaryngology are: Dr. E. N. Broyles, Baltimore; Dr. H. P. House, Los Angeles; Dr. W. J. McNally, Montreal, Canada; Dr. Dorothy Wolff, and Dr. D. Woodman, New York City.



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